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ABSTRACT

This report presents a general framework for studying the acquisition and cognitive representation of biomedical concepts and analyzing the nature and development of misconceptions. The central approach of the report is a selective and highly concentrated analysis of the true nature of clusters of complex concepts and the manner in which they are understood by learners. The report finds a widespread tendency for medical students to develop significant errors in conceptual understanding, including specific conceptual misunderstandings and maladaptive biases in the thought processes used in dealing with complex concepts. The report notes particularly the tendency for misconceptions to compound each other within a general climate of oversimplification, producing large areas of entrenched misunderstanding. The report considers in detail the factors contributing to the acquisition and maintenance of the fallacy concerning the causal basis of congestive heart failure at the level of the muscle cell. The report concludes with a discussion of the implications of the findings for learning and instruction. (Nineteen figures are included, and 39 references and 6 footnotes are appended.) (Author/RS)

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Technical Report No. 440

THE NATURE OF CONCEPTUAL UNDERSTANDING IN BIOMEDICINE: THE DEEP STRUCTURE OF COMPLEX IDEAS AND THE DEVELOPMENT OF MISCONCEPTIONS

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Abstract

A general framework for studying the acquisition and cognitive representation of biomedical concepts is presented. This framework is also applied in analyzing the nature and development of misconceptions. Central to our approach is the selective and highly concentrated analysis of clusters of complex concepts, both as to their true nature and the manner in which they are understood by learners. What we find in these analyses is a widespread tendency for medical students to develop significant errors in conceptual understanding. These errors include specific misunderstandings of concepts and, more generally, maladaptive biases in the thought processes that are brought to bear in dealing with conceptual complexity. Particularly noteworthy is our observation of an insidious tendency for misconceptions to compound each other within a general climate of oversimplification, producing large-scale, mutually reinforcing, and durably entrenched areas of faulty understanding. One such widely held fallacy concerns the causal basis of congestive heart failure at the level of the muscle cell. This fallacy and factors contributing to its acquisition and maintenance in belief are discussed in considerable detail in the paper. The implications of our findings for learning and instruction are discussed.

THE NATURE OF CONCEPTUAL UNDERSTANDING IN BIOMEDICINE: THE DEEP STRUCTURE OF COMPLEX IDEAS AND THE DEVELOPMENT OF MISCONCEPTIONS

In our studies of the nature and developmental patterns of a variety of biomedical misunderstandings (Spiro, Feltovich & Coulson, 1988; Spiro, Feltovich, Coulson & Anderson, in press; Feltovich, Coulson & Spiro, 1986), we have observed 3 important commonalities:

1. *Multiplicity*. Many influences contribute to the acquisition and maintenance of misconceptions, some of which are associated with the learner, some with the educational process, and some even with the practices of biomedical science research.

2. *Interdependency*. Overall misconceptions can be represented as *reciprocating networks* of faulty component ideas which mutually bolster each other and, in turn, support the overall misconception.

3. *Oversimplification* of complex biomedical phenomena and concepts appears to be a major force in the acquisition and maintenance of misconceptions.

Thus, what may seem at first to be a simple misconception, easily describable in a single sentence, will turn out in fact to have numerous complexly interrelated layers of underlying meaning. It is in this sense that we refer to a *deep structure* of ideas. As will be made clear later, these features would likely go unnoticed without a concentrated analysis of the complexities of individual concepts and of the finer threads of relatedness that exist among neighboring concepts.

The paper is organized into three main sections. In the first part of the paper we highlight the central features of our approach and the advantages that accrue from its application. An in-depth analysis of the structure and genesis of the heart failure fallacy in the second part of the paper illustrates more specifically the intricate and convoluted patterns we have found in the development of biomedical misconceptions. Finally, we discuss the implications of our findings for learning and instruction, and for the practice of medical education.

THE CONCENTRATED STUDY OF CONCEPTS AND CONCEPTUAL FAMILIES IN ADVANCED KNOWLEDGE ACQUISITION

Our approach eschews broad but superficial coverage of large numbers of biomedical concepts in favor of in-depth coverage of small sets of important and complex concepts. This decision is motivated by our interest in advanced knowledge acquisition, in how people come eventually (if ever) to understand and apply complex material well. The ideas of interest are ones that are intellectually challenging, ones that require much time and much effort to master; alternatively, they are ones for which, despite effort, understanding may never progress very far for various reasons (e.g., because a particular conceptualization acquired early in learning blocks progress). It is with regard to advanced knowledge acquisition for complex material that current theories of learning are most deficient and current educational methods least effective. In particular, although much has been described about experts and novices (e.g., Chi, Feltovich & Glaser, 1981; Larkin, McDermott, Simon & Simon, 1980), little is known about the *acquisition* of the advanced understandings found in expertise or about the best educational methods for fostering them (but see Collins, Brown & Newman, in press; Spiro, Vispoel, Schmitz, Samarapungavan & Boerger, 1987). Because the kinds of ideas we are interested in *are* difficult and complex and because the processes of learning and understanding them are also complex, and not well understood, the process of *studying* these matters must be detailed and comprehensive.

We believe that being able to deal with complexity is essential if those who are to use complex knowledge, for example, medical practitioners, are to be able to respond effectively to deviations from routine situations that require flexible and adaptive thought. The perspective of advanced knowledge

acquisition and understanding, with its emphasis on the psychological management of complexity, is discussed in the first subsection below.

A second issue that arises from our approach concerns *concept selection*. Because we deal with a restricted set of concepts, we must ensure that those that we do address are important ones, central to the development of biomedical expertise, and ones that are in need of study because they present problems to learners. Our approach to concept selection, based on concept "nominations" by medical teachers and practitioners, is described in the Methods Section.

In the section devoted to the kinds of generality derivable from concentrated conceptual analysis we discuss some of the important advantages that accrue from our approach. Besides the practical benefits that result from the study of concepts suggested by practitioners and teachers as being important and difficult, several kinds of general implications are derived as well. The results of our in-depth analyses possess several kinds of *generality*, including: themes related to content knowledge structure; themes of cognitive representation (in response to cognitive challenges of complexity); themes in the presuppositional, prefigurative schemes that are brought to bear on complex ideas; biases in the management of conceptual complexity; and patterns of misconception *development* that *recur* in different conceptual families, including two types of conceptual compounding. These forms of generality are observed both for incorrect knowledge about individual concepts and for higher-order misconceptions that evolve because of compoundings of several component misconceptions.

Complexity and Advanced Knowledge Acquisition

Certain aspects of our stance with regard to complexity are worth noting at the outset. These concern what we mean by complexity and what we argue is the appropriate place of complexity and the reaction to it in learning and instruction, especially at more advanced levels of knowledge acquisition in a subject area.

Aspects of Conceptual Complexity and Domain Ill-Structuredness

Concepts can be difficult and complex for a variety of reasons. In general they are difficult because they make unusual demands on cognition as compared with more mundane cognitive practices or abilities. There are at least four categories of demand that make a concept hard:

1. *Unusual demands on working memory*. Included here are large numbers of nested steps or goals that must be managed, or large numbers of variables or simultaneous processes that must be reconciled--aspects of multidimensionality.

2. *Unusual demands on formal representation*. Two aspects are important here. One is the degree of abstraction necessary for understanding--the extent to which the concept includes components that are less concrete (e.g., ideas of *rate* or *acceleration* of flow). Another demand involves the semantic distance between concepts and their formal symbolic representations (e.g., as in equations). The number "2" and the concept of *two* that it represents are fairly close. In contrast, the concept of *stroke volume of the heart* and the "SV" that appears in equations involving stroke volume are quite distant. Coordination of the constraints on the symbolic formalization and the (often quite different) constraints on the real *referents* of the symbols can cause great difficulty.[1]

3. *Unusual demands on intuition or prior knowledge*. Concepts can be at odds with ostensibly related prior experience (so that, for instance, importation of seemingly relevant analogies is detrimental), or they can be counterintuitive or discrepant with common sense.

4. *Unusual demands on notions of regularity.* Concepts can be ill-structured: They can be highly variable in their application, requiring tailoring to context, recognition of numerous exceptions, the ability to deal with substantial "grey areas," and so on. Concepts can be highly dependent for their understanding on other concepts they tend to co-occur with and interact with, requiring understanding of elements of reciprocation among families of related concepts.

As will be discussed, characteristics such as these cause problems partly because the demands they pose are at odds with more typical cognitive modes and instructional practices that may be adequate and appropriate for more well-structured or simpler ideas.

The Perspective of Advanced Knowledge Acquisition

Any content area will have facets of well-structured simplicity. These facets are dealt with quite well by existing theories of learning, instruction, and knowledge representation, and they will not concern us here (see Spiro, 1980; Spiro et al., 1987, for reviews). Other facets will be complex and ill-structured. Often, when complexity is encountered in instructional settings, it is transformed into a simpler form to convey an introductory overview of the area to beginners. Complexity is then introduced incrementally, along with directed challenges to earlier "scaffold" models, in order to guide learners toward greater sophistication (e.g., Glaser, 1984). At the early stages of learning, the motivation for strategies of simplification, that of providing nonconfusing immediate access to a subject area, can be appreciated.

However, we have two concerns about this approach. First, we have found that initially simplified approaches often impede the later acquisition of complexity; learners resist what is difficult when they have something simple and cognitively satisfying that has already been learned to fall back on, and early, simpler models provide "lenses" for filtering out ill-fitting aspects of the new material (see Feltovich, Coulson & Spiro, 1986; Spiro et al., in press). Second, we have also observed that the tendency to reduce complexity early is carried over as a kind of habit of thought or instruction to more advanced stages of knowledge acquisition; that is, the reductive modes of learning and instruction applied in initiation to a topic are also employed in attempting to attain a deeper, more sophisticated understanding.

Accordingly, we distinguish *advanced knowledge acquisition* from the kind of learning and instruction frequently advocated for beginners in a subject area (Spiro et al., in press). For example, it may be reasonable to consider the primary goals of instruction at early stages of learning to be that students avoid confusion and not be daunted. The perspective of advanced knowledge acquisition, on the other hand, assigns the highest priority to getting the concepts *right*, even if some difficulty is part of the cost (of course, difficulty should be reduced as much as possible, without sacrificing the *integrity* of the concepts).

Another factor that differentiates advanced knowledge acquisition from standard practices of beginner-level instruction is the nature of assessment: What it is that is considered appropriate and satisfactory evidence of learning. In the beginner modes, reproductive or imitative *memory* for key facts and definitions is the most frequently employed learning criterion. From the perspective of advanced knowledge acquisition, demonstrations of learning involve the novel *use*, *application*, or *transfer* of explicitly taught material (subject, of course, to adequate memory for *prerequisite* information, for the rudiments of a subject area). *Memory for* material that has been taught is not the same as *learning from* instruction (Spiro, 1980). In fact, it seems likely that those factors that promote accurate memory (e.g., tightly compartmentalized, insular mental representation) are *antithetical* to the development of usable/applicable knowledge (Spiro, 1977, 1980; Spiro et al., 1987). In one recent study of students who had just completed a cardiovascular curricular block, we found no positive correlations between performance on memory-type tests and either tests of deep inferential understanding of course concepts or applications of those concepts in clinical tasks (Coulson, Feltovich & Spiro, 1987).

It is important to emphasize that there is no clear split between early and advanced knowledge acquisition--the course of the latter is highly dependent on the former. Understanding is a process in which what is learned about a topic at one time will affect the kinds and levels of understanding that can be achieved on that topic at a later time. The impediments and, possibly, limits to the level of understanding that appear to derive from the strategy of artificially reducing inherent complexity in early learning raise concerns about this strategy. If the goal, ultimately, is deep understanding (ultimately "getting it right"), then the desirability in some situations of fundamentally different approaches to early learning is raised. Alternatives might include exposing students to the full complexity of ideas from the beginning, recognizing that their initial feelings of mastery, accomplishment, and satisfaction may suffer temporarily, but that their horizons of understanding may be greater. Another alternative, discussed at the end of this chapter, involves the instructional use of relatively simple and understandable pedagogical components, but with a guidance system that at every step of learning highlights the limitations and misleading aspects of these components, as well as the linkages and sources of mutual embellishment among them.

The distinctions we have drawn are important because one of our central claims about advanced knowledge acquisition for complex concepts is that the processes of learning, instruction, and mental representation entailed are often diametrically opposed to those commonly found in introductory learning. For example, in examining the heart failure misconception later in the chapter, the same biases found to be maladaptively reductive in the development of misconceptions would appear to be a model set of familiar prescriptions for teaching and learning (and even scientific theory building) in the context of typical introductory learning or in a simple and well-structured domain. What in one situation might be taken to be a goal, in the other is a main obstacle to goal attainment.

Methodology for the Empirical Selection of Concepts for Concentrated Study

An approach such as ours, which focuses in detail on students' learning and understanding of a small number of concepts, requires that the concepts chosen for study be well selected. In order to support our goals, the concepts should meet three criteria: (a) they must be *perceived* by the medical community as important to the practice of medicine; (b) they must *be* important to the practice of medicine; and (c) they should be *difficult* for students (who eventually become medical practitioners) to learn, understand, and apply.

Perceptions of importance matter, if only for pragmatic reasons having to do with potential receptiveness to our findings. It would be discouraging, after much work, to receive a response of "So what?" because a topic we have chosen is viewed as unimportant. *Actual* importance has theoretical significance. One of our goals is to be able to trace the effects of the learning and understanding of basic science ideas into clinical practice. These links can be subtle and circuitous, and our chances of being able to trace impact are greater for the important concepts. These concepts are ones that are critical as building blocks for understanding others, that apply in many ostensibly different circumstances, or that interlock greatly with other concepts. *Difficulty* matters for reasons of potential educational usefulness of our work; difficult ideas, of course, are the most problematic for teachers and students. Perhaps most importantly, it is the most difficult ideas that provide the best opportunity for studying advanced knowledge acquisition, how people adapt to and manage conceptual complexity.

For all of these reasons, we have adopted an *empirical* approach to choosing concepts that relies heavily on the judgments of medical teachers and practitioners. There is a trust that such practitioners have good intuitions regarding the concepts that are both difficult *and* important to the practice of medicine. We are studying concepts nominated by medical school faculty, both basic scientists and clinical faculty (many of whom also practice as physicians). This was done initially through polling of faculty at our own medical school and of a sample of community physicians, and has continued in a survey of all medical schools in the United States and Canada (Dawson-Saunders, Feltovich, Coulson & Steward, 1987). In the survey, faculty are asked to nominate concepts that meet the criteria mentioned earlier: Importance to the practice of medicine and, in view of teaching experience, chronic

difficulty for students in understanding and application. Students' learning and understanding of concepts chosen in this way are then investigated in our laboratory.[2]

Kinds of Generality Derivable from Concentrated Conceptual Analysis

Obviously, there is a *prima facie* medical relevance to studying concepts judged by the medical community (in our survey) to be important and to have wide scope of application within medical practice. In addition, however, findings from the concentrated study of individual concepts have several other kinds of general relevance. We now sketch the various ways that what is learned from a close conceptual analysis has more general implications.

Generality Due to the Cross-Contextual Applicability of Concepts: Content Themes

The same basic knowledge and principles involved in one context may apply in other contexts. For example, the factors that contribute to opposition (impedance) to the flow of blood--resistance related to vessel diameter and blood viscosity, compliance having to do with vessel diameter and the stretchiness of vessels, and inertance having to do with vessel diameter and the mass or density of the flowing material--are also operative in pulmonary air flow. The latter two factors are particularly germane in flow systems in which the flowing material undergoes cycles of acceleration and deceleration (alternating current circuits) as happens in both the cardiovascular and pulmonary systems. Correct understanding in either system, of accelerative effects as they relate to oppositional factors, will yield beneficial transfer to the other system (and incorrect learning will produce negative transfer).

Generality in Types of Cognitive Challenge: Representational Themes

Phenomena or concepts (which themselves represent phenomena) must be mentally represented. A mental representation captures some aspects of what is being represented but may omit others or construe aspects in various alternative ways. Cognitive representations can vary in their fidelity to what is represented, and one of the things that determines the use of one representation over another is the cognitive challenge or difficulty it imposes (see Siegler & Klahr, 1982).

We often find that the mental representations used by individuals show a consistent pattern of response to cognitive challenge. The same cognitive process demands that make one concept difficult are often found to make other, ostensibly different, concepts difficult. An example is the representation of phenomena involving numerous processes or variables. One of the things that makes understanding of opposition to blood flow (cardiovascular impedance) difficult is the need to reconcile simultaneously the effects of resistance, compliance, and inertance (the latter two through their contributions to reactance), compounded by their interactions with heart rate, blood density, blood viscosity, and various other factors. (Reconciling these same numerous variables is, of course, an important challenge in understanding pulmonary air flow.) In a completely different system, discerning the interactions among numerous variables and simultaneous processes is a major cognitive demand that makes the understanding of acid/base and electrolyte balance difficult. Anticipating a general issue that will be taken up in the section on "Themes of Complexity-Reduction," a response of students in both the cardiac and acid/base domains is to under-dimensionalize: The phenomena are represented in ways that constrain them to subsets of the operative variables, rather than dealing with the complex interactions. With regard to oppositional effects in blood flow, the numerous different factors are interpreted in terms of, or as being analogous to, resistance (Feltovich, Coulson & Spiro, 1986).

Another example, involving a different kind of cognitive challenge, is the representation of rates, as is necessary in understanding blood flow as a *rate* of flow (rate of change of position of volume). Such understanding requires the representation of a ratio of differences and poses substantial difficulty for students (even before consideration of rates, which is critical, for example, in understanding accelerative effects). A common student response is to represent *rate* of blood flow as blood *volume*.

This representation is equivalent to treating blood flow as its mathematical integration. Understanding of rate is important in any circumstance in which any kind of material flows, so that a finding of rate-related misrepresentation in one instance has general ramifications.

Generality in the Importation of Explanatory Models: Themes of Field Prefiguration

Prior to selecting the specific cognitive representation that will be applied to a phenomenon or concept, and constraining the nature of that representation, a much more fundamental and often unnoticed issue has often already been decided: What general form should an explanation take? Questions of this sort draw heavily on individuals' or disciplines' beliefs about the fundamental nature of the phenomena they deal with. A prefigurative scheme is a kind of "world view," a set of (often tacit) presuppositions about the fundamental nature of the world (or some circumscribed domain) and about what constitutes legitimate evidence and explanation. Prefigurative schemes are subtler, although perhaps more powerful in their influence on thought, than conceptual content or even its particular cognitive representation (discussed above). Both of the latter, in a sense, refer to things one thinks *about*; in contrast a prefigurative scheme is something one thinks *in terms of* (cf. Bransford, Nitsch & Franks, 1977), a kind of lens that one sees with and that at the same time determines what is excluded from view. Such schemes resemble what have been characterized within science as "paradigms" (Kuhn, 1971). Philosophers of science may study and be aware of alternative paradigms held by the scientific practitioners of a time, but the practitioners themselves function *within* them (*tacit* knowledge); similarly for the implicit paradigms that guide the thought of a medical student.

The notion of a prefigurative scheme is perhaps most easily illustrated in the area of history. There are a variety of established explanatory models that are utilized in historical accounts (cf. White, 1973). Some historical explanations will presuppose a historical *mechanism*, with one event causing another, within chains of cause and effect sequences of events. Other historians will import the model of an *organism*, seeking out some teleological principle of historical development that unites many diverse historical events, but not in a cause-and-effect manner; the historical meaning of an event is treated as more than the sum of its parts or their effects. Still other historians will provide historical accounts that resemble *networks*, with an explanation accumulating as a function of the compounding of multiple interrelationships across the sectors of historical space. Once the metaphor of a mechanism, or an organism, or a network has been adopted as an underlying model, the nature of an explanation, the kinds of phenomena to be selectively focused on, and other important issues have to a great degree already been determined (Pepper, 1942).

Such broad-scale points of view can be applied to biomedical phenomena as well. For example, under a mechanistic scheme, in understanding or explaining biological processes one is more likely to look for causal agents and acts, and to decompose processes into pieces, steps, and causal chains. Under a more organicistic view, the process as a whole will be taken to have primacy over its parts; the function of the process in supporting some larger objective of the organism (e.g., homeostasis) provides guiding principles for the functioning of components, and analytic decomposition is accomplished only at some loss.

When prefigurative schemes are engaged, they have at least three kinds of effects. First, they affect thinking, for example, by affecting what from a situation will be represented cognitively and what will be excluded (or just not noticed). Second, they provide general rules for what constitutes legitimate explanation of, or evidence for, what is going on in a situation. Third, they reflect fundamental beliefs about the *nature* of the world and its phenomena.

Prefigurative schemes can be applied at many levels. At the most encompassing level are ontological schemes of the sort we have been discussing. These address such basic issues as the relationship of parts to wholes. On a still very broad scale, but more concrete, are schemes like the geocentric theory of the universe. Broad-scope scientific theories, such as Newtonian mechanics, also function as lenses

with their own explanatory schemes, modes of evidence, and so on. Schemes for the interpretation of more localized phenomena can likewise come to capture a field and its practitioners in such a pervasive way that they become presumptive, tacit, and prescriptive. Almost as second nature, they come to *be* the phenomena they were originally designed to characterize. With regard to the heart failure misconception to be discussed, the Sliding Filament Theory of muscle contraction will be seen to play a role of this sort.

The discussion thus far of prefigurative schemes should not be taken as implying that only one such scheme can be held or applied by a person at the same time; and, we do not wish to suggest that there is some best lens. For one phenomenon, one scheme will be better, for another phenomenon, another scheme will be better, and multiple schemes applied to the same phenomenon may compensate for the way each single scheme selectively accentuates particular aspects. Rather, we simply wish to suggest the existence of schemes that prefigure cognition and to discuss something of their nature and consequences for cognition. Misconceptions can result from the engagement of a prefigurative scheme that is faulty, inappropriate, or inadequate (e.g., one whose selection of aspects of a phenomenon to be understood is ill-fit to that phenomenon). Also, the engagement of prefigurative schemes can produce errors in systematic *ways* (e.g., misunderstanding of continuous aspects of processes by the importation of a mechanistic scheme that segments those processes).

These commonalities in the ways that a prefigurative scheme can induce misconceptions (i.e., prefigurative themes) are yet another aspect of generality that can accrue from the in-depth study of concepts. Concentrated conceptual analysis can reveal the *underlying* model that is guiding the more specific aspects of a concept's representation. By close analysis of several concepts, patterns of misconception development induced by prefigurative schemes can be identified.

Generality of Cognitive Biases in the Management of Difficult Concepts: Themes of Complexity Reduction

So far we have seen three kinds of generalization associated with the close analysis of sets of concepts: Content themes, representational themes, and prefigurative themes. In this section we show that all of these themes are associated with a directional bias in the way they are cognitively instantiated. The bias in each case involves the reduction of complexity.

"It costs energy to make surfaces that are rough" (Gleick, 1987). A sphere is the lowest energy configuration that a soap or water bubble can assume, and it is the one toward which its surface forces tend; to make a bubble assume any other configuration requires the input of energy. The preceding quote is from a discussion of the formation of a snowflake as a process that at all stages must overcome the surface tension of water, which would tend naturally to make the snowflake round. Everywhere in nature there are strong forces tending toward simplicity. Recent theories of information processing and knowledge representation (Smolensky, 1986) have suggested a similar tendency for cognitive processes and interpretations to settle into the lowest energy configurations, in a sense the ones that can be assumed with the least effort. Hence, it should come as no real surprise that there are such simplifying forces playing a role in the understanding of complex concepts; as in the making of an exquisite snowflake, intricate understanding may also have to oppose such "reductive forces."

Thus, another area of generality that emerges from the study of concepts involves the nature of and principles governing these kinds of cognitive reductions, their ramifications for misconception, and, ultimately, insight into ways they can be overcome in advanced knowledge acquisition. The concepts we have chosen to study *are* difficult and complex. Close conceptual analyses reveal the strategies (intentional and unintentional) that students and teachers adopt in the face of that complexity. Students are sometimes able to cope with difficult concepts without trivializing them, but, unfortunately, this does not appear to happen very often. Instead, there is a proclivity toward the strategic mismanagement of complexity, involving various forms of *oversimplification*. Close analysis of conceptual understanding in a variety of biomedical domains has revealed numerous misconceptions

that have in common some form of convenient and spuriously supportable oversimplification of a complex concept or phenomenon. We have referred to these comfortable oversimplifications as *seductive reductions* (Spiro, Feltovich & Coulson, 1988). Just considering the use of analogy in learning, we have observed biomedical misconceptions resulting from eight different varieties of spurious reduction of a new concept to an analogical source (Spiro et al., in press).

The misconceptions that we have characterized as seductive reductions result from the application of a large set of beliefs, motives, and cognitive operations that we refer to individually as reductive biases and collectively as *The Reductive Bias* (Coulson, Feltovich & Spiro, 1986; Spiro, Feltovich & Coulson, 1988). Many of these reductive biases will be illustrated in the context of the heart failure misconception. For now, we will give a few illustrative examples, concerning, first, reductive biases in conceptual understanding and, second, such biases in learning. It will be noticed that both of these kinds of reductive biases can operate in connection with all three kinds of themes we have discussed: Contentive, representational, and prefigurative (see the subsection below, "Realms of Reduction").

Conceptual biases involve systematic ways of spuriously reducing complexity in understanding. (Note that despite some similarities, conceptual biases are not the same thing as biases in judgment and decision making; see Elstein in press, Kahneman, Slovic, & Tversky, 1982, for discussion of the latter literature.) For example, there is a tendency in understanding the cardiovascular system to eschew dynamic (continuous, changing, etc.) interpretations in favor of a more static view, as when students equate blood flow with blood volume (*static bias*--a conceptual bias applied to a representational theme). Other examples of conceptual biases include the following:

- *Step-wise bias*. In another conceptual bias involving representation, continuous processes are broken down into discrete steps, with loss of properties that exist at the holistic level. This is often done by students in representing the continuous flow of blood as a sequence of steps, causing them, for example, to misunderstand *relationships* between outflow and inflow to the heart.

- *External agent bias*. Intrinsic characteristics of entities or processes are attributed instead to external influences--as when vascular compliance is taken by students to be the vessel's ability to respond to orders from the nervous system. This confuses external mechanistic forces with inherent, internal processes of development (organic processes), a common consequence of applying mechanistic *prefigurative* schemes.

- *Prior analogy bias*. New concepts are interpreted through already held (often simpler) models, often imported from extra-instructional experience (e.g., the cardiovascular system is interpreted as being too much like household plumbing). This is a bias toward assuming more *contentive* similarity than is warranted.

- *Common connotation bias*. Technical terms are interpreted according to their everyday, common language meaning (e.g., the different types of erroneous models of vascular compliance we see correspond to various dictionary definitions of "compliance," as in the example above--also see Feltovich, Coulson & Spiro, 1986).

- *Restriction of scope bias*. General physiological principles are thought only to apply in specific instances (e.g., within conditions of pathology). This bias, in caricature, might support the proposition that the laws of gravity hold only on earth. In the biomedical arena, vascular resistance is taken by some students to be a property only of small blood vessels.

Acquisition biases are modes of addressing complex ideas during learning that attempt to make the ideas more tractable. Examples include *under-dimensioning--a representational* approach of teaching or learning multivariate phenomena one dimension at a time, with a goal toward eventual dimensional reassembly; *atomization and extirpation--a prefigurative* bias that extracts and isolates components from a multi-component system, with the assumption that their behavior in isolation will faithfully reflect their behavior in context; and *sanitizing--a contentive* bias that involves focusing (in learning and instruction) on the clearest, cleanest instances of a concept, those most insulated from contextual effects, with the idea the these will be representative of, or will be a"bridge" to, the messy contextual exceptions.

Note that some acquisition biases directly parallel and reinforce conceptual biases. An example is the approach to learning about continuous processes that involves breaking them into component processes with sequential steps and effects (as is done routinely, for instance, in the learning/teaching of the cardiac cycle--paralleling the step-wise bias).

Realms of reduction. The Reductive Bias applies to all three kinds of themes that we have discussed--those involving content, representation and prefiguration--and in each of these areas functions as a kind of "selector" that opts for simpler over more complex modes of interpretation.

With regard to *content*, the most common result of the application of The Reductive Bias involves treating things, or aspects of things, as being more similar or more stable in their characteristics than they are. (A consequence of this similarity bias is a tendency to produce overgeneralizations.) Forms of this contentive reductive bias appear in the use of analogy (Spiro et al., in press), e.g., *analogy treated as isomorphism*: analogy is treated as being the same relations, based on the same underlying mechanisms. Other examples include the following: *extension of attributes*--if A is like B with regard to attribute X, then A will be like B with regard to attributes Y, Z, and so on;[4] *homogeneity of components*--explanations that account for one component of a system will account for others of the same nominal type, for example, "a muscle is a muscle," whether it pumps blood (cardiac) or "pumps iron" (skeletal); and *reduction of technical meaning to common connotation*, discussed earlier.

Many of the options available for cognitive *representation* can, likewise, be seen to span a simpler-to-more-complex dimension. Should a concept or phenomenon be represented cognitively as: unidimensional vs. multidimensional; static vs. dynamic; compartmentalized vs. interconnected; linear vs. non-linear; continuous vs. discrete; and so on? The operation of The Reductive Bias selects for the simpler pole among representational options, even when it is not appropriate.

There are ways that *prefigurative schemes* (lenses) can be characterized so that they too can be seen to vary in complexity. Such a characterization has been proposed by Pepper (1942) and involves two bipolar dimensions of classification: analytic-synthetic and dispersive-integrative. For analytic schemes, parts are the facts of a phenomenon, and combinatorial interaction of parts and synthesis are derivative; in contrast, for synthetic schemes wholes and emergent properties are basic and decomposition is derivative and reductive of the phenomenon. While this first dimension deals with the relative primacy of parts and wholes, the second addresses inherent orderliness. Dispersive schemes accentuate the irregularity and ill-structuredness of events, while integrative schemes presuppose more coherence, orderliness, classifiability, and so forth.

The Reductive Bias leads to "pole migration" with regard to these schemes, that is, to the adoption of those prefigurative schemes that are simpler and easier to manage cognitively--the analytic and the integrative. This manifests itself in reductive biases such as *atomization and extirpation*, the presupposition that "parts" extracted from context will faithfully reflect their properties and dynamics of operation in full context (analytic), and *uniformity of explanation*, the presupposition that complex processes are *really* governed by some single principle or mechanism (integrative).

The adoption of the *simpler* schemes promotes a kind of double jeopardy. First, it bolsters the application of *single* lenses or schemes to phenomena--a uniformity of 'lens' application. For example, dispersive schemes are antithetical to the notion that any uniform explanation can be applied everywhere, while this uniformity is the *forte* of integrative schemes. In turn, when analytic or integrative schemes are adopted, the world is then selectively interpreted that way, filtering aspects that fall "off-line," and bolstering the adoption of the reductive schemes in the first place. An integrative scheme promotes single lens application--single lens application bolsters the apparent efficacy of the integrative scheme. A danger is the belief that the scheme is accounting for more of the variance in a phenomenon than it actually is. An example that plays a big part in the heart failure misconception is the assumption that the Sliding Filament Theory accounts for all muscle cell contractile dynamics, when, in fact, it accounts for only some.

Why are reductive cognitive modes of all kinds so readily adopted? One reason has to do with cognitive ease. It is easier to think that all instances of the same nominal concept, for example, *compliance*, are the same or bear considerable similarity (*contentive* reduction). It is easier to represent continuities in terms of components and steps (*representational* reduction). It is easier to deal with a single principle from which an entire complex phenomenon "spins out" than to deal with numerous, more localized principles and their interactions (integrative *prefigurative* reduction).

Furthermore, reductive tendencies are reinforced by many educational practices, particularly those associated with introductory teaching and learning. We have argued that the educational process in general is riddled by an implicit and unintended *conspiracy of convenience* (Spiro et al., 1987) to treat complex subject matter as simple. This makes it easier for students to learn, for teachers to teach, for textbook authors to write, and for testers to construct and grade their tests. Thus, besides making a difficult situation easier for everybody, The Reductive Bias finds the grounds for its acceptance already prepared, because the elements of this conspiracy have already been established in what are commonly taken to be effective modes of teaching and learning: highlights, conceptual "coat racks," sanitizing (not confusing students with all those exceptions), glossed exposure, etc.

Adding to the problem is that for some circumstances and some kinds of learning, such reductive maneuvers may be appropriate and effective. Much that has been pointed to negatively here is often taken as efficient and effective cognitive processing (e.g., *extension of attributes* as "default assignment"--see Footnote 2), effective representation (e.g., *decomposition* of a complex problem into components), or even standard canons of science (e.g., investigating the dynamics of complex phenomena by varying one dimension at a time). Such common practices are appropriate where they are appropriate--generally with regard to well-formed concepts and well-structured domains. However, in more complex and ill-structured domains these very same practices are problems, not principles. One might argue that what makes truly difficult concepts hard is that they fall "off-line" in ways that undermine mundane cognitive and investigative tools; if they were routine and usual, they would not be hard.

Generality of Patterns of Development of Higher-Order Misconceptions: Themes of Idea-Compounding and Spreading Misconception

Although we focus at first on individual concepts, the fact that many of the concepts we choose form groups of conceptual clusters or families allows patterns of higher-order conceptual representation to emerge. We have discussed how our approach to the close analysis of individual concepts informs both about the detailed nature of the understanding of those important concepts and, by allowing observation of themes that recur across individual concepts, about certain general principles and problems of conceptual understanding. We now want to demonstrate a different kind of benefit: By the concentrated study of the individual concepts in a conceptual family, otherwise hidden patterns become evident in which *misconceptions compound each other* and thereby contribute to higher-order misconceptions. These higher-order misconceptions may be even more seductively entrenched than the local ones. *Spreading activation* has been a popular topic in recent cognitive science (Anderson,

1983; Collins & Loftus, 1975) by describing the processes by which ideas compound each other, we introduce the notion of *spreading misconception*.

Such higher-order misconceptions take a variety of forms, of which we will illustrate two. One involves *compounding*: Misinterpretations of fundamental ideas can cohere in systematic ways such that belief in one makes belief in others easier. Consider the following example (see Feltovich, Coulson & Spiro, 1986 for a detailed treatment). The effect of vascular compliance (a factor related to the stretchiness of vessels) in blood circulation is often interpreted by students as a kind of "surrender," as enabling vessels to "give way" and accommodate increases in blood flow. This is in contrast to the *active* role of compliance (and elasticity, its inverse) in promoting the movement of blood. Believing compliance is primarily accommodative makes it easier to believe that veins, highly compliant vessels, are reservoirs, places for holding blood, as opposed to active participants in ongoing dynamic flow. And, *reciprocally*, the latter belief bolsters the former one. Furthermore, pressure, in relation to blood flow, is interpreted as hydrostatic "bucket" pressure--and not in terms of dynamic gradients--consistent with the first two beliefs. Downplaying the active role of compliance bolsters belief that impedance (opposition to blood flow) is the same as resistance, ignoring or subsuming effects of compliant and inertial reactance (which, unlike resistance, are totally dependent on dynamic aspects of blood flow, as they interact with compliant properties of blood vessels and other factors). This restriction of oppositional effects to resistance (as well as most of the interpretations listed above) is consistent with a view of the cardiovascular system as a "direct current" as opposed to "alternating current" circuit--which is bolstered by the non-recognition of accelerative properties of blood circulation due to pulsatile (continuously changing) pressure. It is also consistent with a view of the vasculature (e.g., veins) as simply accommodating active effects from the heart. And so on.

The compounding and mutual bolstering of these individual interpretive units leads to a second kind of higher-order conceptual effect--*pervasive coloration*. All conspire to promote a *passive* coloration in the "view" of the cardiovascular system, especially regarding the vasculature itself. Once such colorative effects emerge, they can then feed back and shade further learning and interpretation.

The compounding of misconception is aided, in part, by the reduction of phenomena to the simplified poles, for example, the representation of pressure statically and the contentive reduction of all impedance factors to resistance. Convergent shearing away of complicating factors provides the ground for individual factors, thus stripped, to complement each other and to compound. In turn, the broad colorations that then pervade the overall image of a phenomenon (e.g., the "passive" cardiovascular system) can turn back and reinforce thinking about individual concepts that way.

Generality of Learning Scenarios that Cause Conceptual Error: The "Anatomy" of Misconception and its Development

We have discussed the implications of close conceptual analysis under four thematic headings: content themes, representational themes, themes of field prefiguration, and themes of complexity-reduction. Each of these four general kinds of cognitive phenomena is associated with its own distinctive form of contribution to misconception and participates in spreading or compounding the development of higher-order misconceptions (a fifth kind of theme). Considering together the first five kinds of generality described in this section, the result of our approach to close conceptual analysis is a detailed *anatomy* of the nature of conceptual understanding and the development of conceptual error, at the levels of the individual concept and conceptual clusters. This includes insight into the nature of misconceptions, their sources and their interrelationships.

The investigation of concepts in detail, but concepts that are themselves interrelated (a cluster approach), can uncover such interdependencies. This cluster approach enables investigation of emergent effects and broader scale misconceptions that would not be possible through the investigation of the constituent concepts in isolation. As the example of the misunderstanding of congestive heart

failure will illustrate, the sources of misunderstanding can be many and can run far below the surface misunderstanding itself. The specific nature and causes of the heart failure misunderstanding to be discussed would not have emerged without our detailed and interrelated probing of such important constituent concepts of cardiovascular function as: *opposition to blood flow* (impedance), which pertains to factors which oppose the flow of blood; *the Frank-Starling cardiac function* and *Guyton's vascular function relationships*, which capture the intrinsic control of cardiovascular flow, through the interlocked regulation of vascular function by the heart and of cardiac function by the vasculature; *cardiac muscle activation and control of contraction*, which pertains to how the heart muscle itself works to produce its pumping action; and to a lesser extent, cardiac hypertrophy, *cardiac electrophysiology*, and *energetic metabolism*.

Only by looking at these interrelated concepts together, following in-depth investigation of each, could the pattern of interlocking misconception that we will present be detected. In other words, a broad and superficial probing of heart failure knowledge would have missed much of the important underlying basis for the misconception. In a related way, broad, superficial or compartmentalized instruction on heart failure, by missing detailed treatments of constituent basic science concepts and their complex interactions, would result in the "climate of oversimplification" that produces misconceptions of the kind we have found.

In the remainder of the paper, the misconception of heart failure is addressed as a more concrete example of the application of frameworks that have been introduced in these early sections. It will be shown how misconceptions interact to bolster each other, compound to yield yet higher-order misconceptions, and align in gross aggregation to produce pervasive colorations--all within an erroneous belief-structure about the basis of congestive heart failure. The various kinds of compounding of interactions suggest a network structure for the overall belief system, a network that yields synergistic strength greater than the component parts. Paralleling the learner's interpretations and modes of thought (related to addressing complexity) that lead to and support misconception, similar reinforcing influences from the instructional process and from some practices of biomedical science are also discussed. It is argued that rather than being external to the cognitive network of misconception and simply contributory, these "external" influences are entwined inseparably within the network itself; they are partly responsible for the component misconceptions that come to make up the network and are partly responsible for the patterns of thought and practice that legitimatize and, hence, help to maintain the network. Learning, educational practice, and laboratory science are seen to be subject to the same reductive biases and, therefore, to be symbiotically enmeshed.

THE "ANATOMY" OF A MISCONCEPTION OF HEART FAILURE

The misconception of heart failure is taken up next, following the introduction of some necessary background information regarding congestive heart failure and cardiac muscle cell function.

Background Regarding Heart Failure

Congestive heart failure is a syndrome or constellation of effects in which the heart loses its effectiveness as a pump and fails to maintain flow rates consistent with the needs of the body. The misconception to be discussed has to do with the dynamics at the subcellular level of the heart muscle that account for heart failure. In order to understand the nature of the misconception, a brief overview of these subcellular dynamics is necessary.

The heart pumps by contracting its myocardial muscle. In heart failure this contraction is inadequate, lacking sufficient force, shortening capacity, or both. Figure 1 depicts the subcellular part of a muscle, the sarcomere, which ultimately produces contraction.

[Insert Figure 1 about here.]

Within each sarcomere are myosin and actin filaments. The sarcomere is the unit between the two vertical bands (Z-bands) depicted in Figure 1 (also depicted in the figure are adjacent sarcomeres; i.e., sarcomeres are aligned in series, abutting at shared Z-bands). The myosin (see Figure 1) is a fixed-length filament that has as components a number of little "arms" (cross-bridges) that ultimately produce contraction. Actin are other fixed-length filaments that are attached to both ends of the sarcomere. These structures, attached to opposite ends of the sarcomere (to the Z-bands), are pulled toward the center of the sarcomere (see Figure 2) during a contraction, shortening the whole sarcomere without substantially changing the length of either (myosin or actin) filament structure. These subcellular structures and dynamics, actin and myosin filaments of fixed length which "slide over" each other during contraction without change in length to either, are components of the Sliding Filament Theory of muscle contraction (Gordon, Huxley & Julian, 1966).

[Insert Figure 2 about here.]

The cross-bridges of the myosin produce the force of contraction. During a contraction, the heads of the cross-bridges bind to available sites on the actin, pull one stroke toward the middle, release, rebind to the next available outward site, pull again and so on, progressively pulling the actin (which is attached to the outward ends of the sarcomere) toward the middle, resulting in shortening of the sarcomere. This process (binding, pulling, releasing, rebinding, pulling, etc.) continues, in the presence of adequate metabolic (energetic) materials available and necessary to drive the process (energy is needed to make the sarcomere "go"), as long as the contractile process remains active.

The *force* of contraction is a product of two main things. The first is *anatomical/mechanical* and involves the degree of alignment of actin surfaces with myosin "arms." One can see that if, somehow, the actin were to get stretched too far outward, some of the arms would have nothing to bind to and force would decline. This is partly due to the constancy of length of both the actin and myosin filaments during contraction, consistent with the Sliding Filament Theory. The second factor affecting contraction force is *activational*. It involves the number of arms that are recruited to participate (to pull) in the contraction (not all need be), and is regulated by the degree of energetic activation of the actin filaments' cross-bridge binding sites. One can envision the actual *number of arms* engaged in the pulling operation being activationally regulated, resulting in degrees of strength of the pull.

Preview of the Misconception

The misconception held by medical students, some medical practitioners, and portrayed in some medical texts holds that heart failure happens because the heart gets too big, stretching component sarcomeres so that actin does not overlap the myosin optimally. This results in fewer potential binding sites, with a corresponding reduction of force of contraction. Hence, of the two main factors affecting force production in a muscle, one anatomical and the other activational, in the misconception the anatomic, mechanical component is invoked, with general neglect of the activational component. In reality, it is the activational mechanisms that are germane to heart failure and the mechanical/anatomic component can play no role. This is discussed after consideration of a few representative statements reflecting the basic misconception given by subjects in our laboratory and in a textbook of cardiology. The quotes from subjects are from responses to open-ended but focused discussion questions (organized by the 'target' concepts, e.g., *muscle activation and control of contraction*, listed in the last section) about the function of the heart and cardiovascular system (see Footnote 1). The first quote is from a second-year medical student (Figure 3); the second, from an established cardiovascular physician (Figure 4); and the third, from a respected and reasonably current textbook of clinical cardiology (Figure 5).

[Insert Figures 3, 4, & 5 about here.]

All of these descriptions portray a common mechanism as explanation for heart failure: If the heart gets too large, then the individual sarcomeres contained within the myocardial muscle of the heart

likewise get stretched; potential binding sites on actin structures are lost due to non-alignment; and hence, the force generated within a pumping stroke of the heart is diminished, producing heart failure. This is a mechanical, anatomical account of heart failure. While this account has a certain seductiveness--it *seems* plausible, and it invokes the usually well-learned Sliding Filament Theory--it is inconsistent with the best available physiological and pathological evidence for the basis of heart failure.[5]

In contrast to this account, the basis of heart failure is activational. The muscle in a failing heart is "sick" muscle, and the basis of this sickness is metabolic and energetic (Katz, 1977); the failing heart does not get energized sufficiently to do its job. While some details of this activational account remain to be specified within laboratory science, the mechanism of heart failure likely involves some combination of actual *damage* to sarcomeres, defects of biochemistry in the activation system itself, altered activity of ion pumps, and perhaps many other defects of muscle chemistry yet to be discovered (Nair, Cutilletta, Zak et a., 1976; also, see Coulson, Feltovich & Spiro, 1986, for additional related references).

A Pervasive and Powerful Misconception

Despite its inappropriateness, the "mechanical overstretching" account appears to be widely held in the medical community. This account has been given in our laboratory by medical students and by some established cardiovascular physicians, and is often described in textbooks. As medical school faculty involved in the normal teaching activities of the school, we have seen this explanation commonly conveyed to students. In our laboratory work with medical students, explanations of heart failure as resulting, exclusively or in part (interspersed with other mechanisms), from mechanical overstretching of muscle fiber were given by 18 of 28 (64%) first- and second-year medical students (first-year: 7 of 14, 50%; second-year: 11 of 14, 79%).[6]

In addition to its pervasiveness, the misconception appears to have a certain kind of insidious *power*. In much of our other research with medical students, we have often been able to trace a direct influence for misconceptions and errors to the primary instructional materials used by the students in their course work (Feltovich, Johnson, Moller & Swanson, 1984). Medical students take their assigned materials seriously; to some extent, "they are what they read." However, there is something about the heart failure misconception that undermines this general rule-of-thumb. The main assigned cardiovascular textbook (Katz, 1977) used by our medical student subjects contains a contemporary and appropriate activational account of heart failure; in the chapter on heart failure there is no mention of overstretching and sarcomere disinterdigitation as a basis for heart failure (although, as we have seen, students may read such accounts in other places). This overriding of primary instructional material emphasizes both the aggregate power of the misconception and the possibly circuitous nature of its influences.

As we have described, heart failure is activational and biochemical. Yet, students believe its basis to be mechanical and anatomical: Our question is, *Why*? We have already suggested that the reasons are many, and, at least in part, circuitous and subtle. In the next section, the question of why students (and others) acquire and maintain the erroneous belief, and the nature of the belief itself are addressed in greater detail.

The Nature, Acquisition, and Maintenance of the Misconception

In this section we investigate why the *mechanical overstretching* misconception of heart failure is acquired by students, the influences that contribute to the robustness of the belief, and the structural form of the misconception itself. As a preview to this section, four general points can be raised:

1. In the misconception of heart failure, dynamics of individual, isolated *skeletal* muscle fibers are mapped to the intact functioning of the whole heart (which, of course, is not skeletal and is a system of *many* fibers).

2. Properties of the whole heart that exist because it *is* a system containing complexes of fibers are neglected within the misconception.

3. The overall misconception contains *component* misconceptions that interact and support each other in reciprocating ways, forming a network. The interlocking nature of these components yields an overall structure, the misconception, that is itself highly robust.

4. Simplification of complex ideas, on the part of the *learner*, the *educational process*, and, in some instances, the *practices of laboratory biomedical science* contribute to the development of the misconception and support its maintenance in belief.

This section is organized around four component misconceptions that are involved in the overall misunderstanding. As overview, these four components are: (a) that the means by which an individual, isolated (skeletal) muscle fiber produces different contracting force at different lengths of stretch (the *length-tension* relationship of muscle fiber, L-T) is solely mechanically and anatomically based; (b) that an individual cardiac muscle fiber is like an individual skeletal fiber; (c) that the increased force that an intact heart (*in vivo*) develops when it is filled with more blood volume (and gets bigger) results from the same mechanisms that enable an individual fiber to produce more force (up to a point) as it is stretched to greater length; and finally, (d) that when a heart becomes too big it loses its ability to generate adequate force, hence fails, for the same reasons that an individual skeletal cell loses its ability to generate tension when it is stretched too far (that is, because of disinterdigitation of actin/myosin filaments). These component ideas are taken up, in turn, along with the introduction of some necessary background for each where necessary. For each component, educational influences toward its development on the part of the learner and the teaching process are discussed, as well as pertinent aspects of laboratory biomedical science.

Component 1: The Entire Length-Tension (L-T) Relationship for an Isolated Skeletal Muscle has an Anatomical Basis (*Length-Tension is Anatomical*)

Background to component 1. The length-tension relationship of a skeletal muscle (see Figure 6) shows the maximum tension that an individual muscle fiber can produce when the fiber is stretched to different lengths. This relationship is determined in the laboratory, using muscle fibers extracted from animals. The fiber is mounted at different lengths in a laboratory preparation, minimally activated to produce a tetanic contraction (the muscle contracts and *holds* its contraction), and the maximum tension produced by the muscle at a given length is recorded. As can be seen in Figure 6, the maximum tension that can be developed rises from low tensions at short lengths to a plateau at intermediate lengths, and with yet further lengthening the ability to generate tension progressively falls.

[Insert Figures 6 & 7 about here.]

The misconception in component 1. The *misconception* held by students is that the differences in maximal tension potentially generated by an isolated skeletal muscle across its entire range of length (the length-tension relationship) are due to mechanical/anatomic factors, as follows. At lengths of fiber corresponding to the plateau, tension is greatest because of optimal alignment of actin and myosin filaments within the sarcomere of the muscle fiber. Force declines at longer lengths (descending limb of Figure 6) because, with stretching, potential binding sites are lost. Force declines at short lengths (the ascending limb) because of some kind of shortening-induced physical impediment to movement of the cross-bridges themselves, or to the physical structures that are moved during a contraction.

This supposition of an anatomical/mechanical basis for the entire range of the length-tension relationship is described in an example protocol from a medical student, given in Figure 7.

What's wrong? The characteristics of the length-tension relationship on its plateau and descending limb can be accounted for by the mechanically based Sliding Filament Theory, but this theory does not apply at all to the ascending limb; while the plateau and descending limb of the L-T are the product of optimal and (degrees of) suboptimal engagement of cross-bridges, the ascending limb (lift-side) is not. The decline in tension by a muscle fiber at short lengths is a matter of diminution of muscle activation. In laboratory science, the activational basis for the ascending limb was revealed through the discovery of an automatic activational shut-down mechanism that engages when a fiber approaches short lengths when contracting; the very existence of short sarcomere lengths inhibits the activation. If means are used to override this shut-down mechanism, so that the fiber can be fully activated, tensions produced at fiber lengths corresponding to those on the ascending limb "spring up" to levels similar to those on the plateau (Jewell, 1977).

Hence, there are dual explanations underlying what is in appearance a fairly symmetric, continuous curve. The existence of this nonuniformity of explanation, within what appears to be such a clean, well-formed relationship, violates what we have identified as a prefigurative preference for uniform, integrative accounts.

Why Do Students Believe Component 1?

In discussing this and other component misconceptions, we will instantiate the framework for conceptual understanding (misunderstanding) developed in the first part of the paper. Various parts of the framework will be instantiated, because the reasons for the misunderstandings are multifaceted. No single factor leads to misconception; rather, partial contributions, each reducing or simplifying the phenomenon in small ways, align in their simplification to yield robust and coherent, but erroneous, belief. These contributing factors involve the student, the educational process, and some practices of the biomedical science laboratory (see Figure 8).

[Insert Figure 8 about here.]

Science. Before discussing the contribution of laboratory science to the students' misconception regarding the length-tension relationship, it is useful to mention the origins of this relationship itself. The classic length-tension relationship of muscle fiber that plays a key role in the misconception of heart failure is based on *skeletal* muscle from the hind-leg of *frogs*. It is interesting to note why this particular muscle was the one used in the late 1800's to establish this relationship. This muscle is large and easy to extract from frogs. Its structure is also conducive to easy mounting in the kinds of laboratory preparations required to determine tension produced at various lengths. Furthermore, the legislation in Britain responsible for the protection of animals in laboratory research at the time the experimentation was conducted did not (and still does not) consider amphibians to be "animals." Hence, for several reasons largely pertaining to *laboratory convenience*, the length-tension relationship of frog skeletal muscle exists as a classical teaching example for muscle function and shades students' ideas about how *heart* muscle works.

As we have noted, the tension developed by a muscle fiber is a function of both anatomy and activation level. Hence, activation is a third variable which modulates the tension developed at any length. In establishing the length-tension relationship within the scientific laboratory, activation was controlled to a single, maximum value. Hence, in this complexity reduction (under-dimensioning) of the response space of tension, the critical activational component is downplayed.

Students. Reductive tendencies on the part of the student also contribute to the idea that the basis for the entire length-tension relationship is mechanical. One is the desire for *uniformity in explanation*. This is reinforced by prescriptions about what good scientific explanation should be, for example,

Occam's Razor, and maxims advocating singularity of explanation within medicine--"never two if one will do."

Another influence on the part of students is an over-reliance on a rowboat and rowing crew analogy (*reductive analogy*) for understanding dynamics of the muscle sarcomere (see the student protocol in Figure 3). This analogy conveys the idea that if the boat were shortened for a fixed number of rowers, the oars could get tangled up, or efficiency could otherwise be reduced by mechanical/anatomical obstructive factors.

Teaching. The mechanical components (related to anatomy) of tension production are easier to teach than the activational components; the mechanism of tension production by the cross-bridges (filaments) is less complicated. It involves only a few key elements (e.g., invariant filament lengths, shortening occurs by relative filament translation) and, especially when misinterpreted, is linear and, hence, easy to represent--the more potential binding sites, the more tension produced. In contrast, the process of activation involves numerous interacting subcellular components and processes (e.g., calcium ion release sites, binding proteins, ion pumps), and the effects are non-uniform (e.g., activation works qualitatively differently at different levels of anatomical extension). Second, as we have seen, neat, easily envisioned and cognitively productive analogies are readily available to aid anatomical understanding (for example, rowing crews and tug-of-war). Finally, details of the physiological activational account are less well understood and are more controversial in some particulars. Options and controversy are difficult to deal with in both teaching and learning. (Anecdotes are widespread that medical students dislike competing or controversial accounts.) Hence, mechanical factors are likely to be stressed to a greater extent in teaching (reflecting, perhaps, yet another reductive bias: *Teach toward the simple*), better explicated, and almost surely more readily understood by the student.

Furthermore, textbook presentations of the length-tension relationship that students see and study depict the relationship at its single maximum activational level (*under-dimensioning*), reflecting the laboratory demonstrations discussed earlier. Three dimensional surfaces are difficult to represent in text.

Implications. The convergence of reductive factors such as those discussed leads to the de-emphasis of activational factors, in favor of mechanical/anatomical factors, in the production of tension (force) by the force-producing unit of a muscle. Playing a large role in this de-emphasis of activation is the overextension, in students' thinking, of the mechanical Sliding Filament Theory to the activationally based ascending limb of the length-tension relationship--thus eliminating altogether the need to consider activational aspects. But the muscle which has been discussed is skeletal muscle--not cardiac muscle--and it is from a fog! What, if anything, does any of this have to do with the heart?

Component 2: Cardiac Muscle is Like Skeletal Muscle: Both Have the Same Length-Tension Relationship (*Cardiac L-T = Skeletal L-T*)

The misconception in component 2 The functional dynamics of *cardiac* muscle fiber are assumed by students to be the same as for skeletal muscle (from the perspective of muscles of the body, an assumption of *homogeneity of components*). This includes the presumption that the two kinds of muscle generate the same kind of length-tension curve. Direct statements of this correspondence are generally not made by students (as it probably has the status of a tacit presuppostion), but the correspondence is indirectly indicated by the description of the classical *skeletal* muscle length-tension relationship in discussions of the way the intact *heart* functions. (See the quote in Figure 9 and also the earlier textbook account in Figure 5 which more overtly equates the two L-T curves in its parenthetical statement.)

[Insert Figure 9 about here.]

What's wrong? Heart muscle is very different from skeletal muscle. Two kinds of operational differences are most pertinent to the present discussion. First, heart muscle fiber produces a very different length-tension curve (see Figure 10, where the cardiac length-tension curve, solid line, is superimposed on the skeletal curve). In the cardiac curve, the potential for the development of tension continues to rise across fiber lengths where tension would fall in a skeletal muscle. In addition, there is *no corresponding cardiac descending limb*. In laboratory preparations used to establish the cardiac length-tension relationship, cardiac fibers are irreparably damaged or break before they can be stretched to lengths corresponding to most of the skeletal descending limb. Second, cardiac and skeletal muscles activate and contract in very different ways. Skeletal muscles contract at maximal activation and produce tetanic contractions; they contract and hold the contraction until release. In contrast, cardiac muscles are always submaximally activated and they twitch, never achieving tetanic contractions.

[Insert Figure 10 about here.]

Why Do Students Believe Component 2?

Science. Although cardiac and skeletal muscle are different in many ways, these differences were artifically minimized in establishing a length-tension relationship for cardiac muscle. Two means of reducing differences are particularly noteworthy (see Figure 11). First, the particular kind of muscle within the heart most similar anatomically to skeletal muscle was used for the demonstration. This is the papillary muscle which controls the action of heart valves. This muscle has a linear structure conducive to mounting in the laboratory and is amenable to a specification of length. It is not a muscle that pumps blood. In contrast, muscle involved in pumping blood, myocardial wall muscle, is *not* conducive to demonstrating a length-tension relationship. Such muscle is difficult to extract, difficult to mount, and is quite irregularly shaped, such that the concept of "length" makes little sense. Second, contrary to the normal submaximal twitch activation of heart muscle, pharmacological means were found to artifically "jack-up" the activation of the heart muscle fiber to produce the tetanic contractions of a length-tension relationship (Gibbs & Loiselle, 1978). This further de-emphasizes the role of variable levels of activation in the production of force by the cardiac muscle. It also obscures the fact that because heart muscle is naturally submaximally activated, large ranges of force production are possible at *any* given (attainable) level of stretch.

Students. Although cardiac and skeletal muscle are different, they are also alike in some ways; for example, they both have sarcomeres containing actin and myosin filaments, force is generated by the binding and pulling of cross-bridges, and, within their respective dynamic ranges of length, more accessible binding sites yield more potential force. Given the similarity of skeletal and cardiac muscle on some of their features, there is an assumption by students of similarity on others (*extension of attributes*). In particular, given the similarities in the two kinds of muscle noted above, it is assumed they should be similar in their activational properties as well (which they are not). In its extreme form this kind of extension would lead to the idea that "all muscles are alike"; findings for any kind of muscle, whether skeletal, heart, papillary, or myocardial, are interchangeable (*homogeneity of components*).

[Insert Figure 11 about here.]

Teaching. The classical *skeletal* muscle length-tension curve is the length-tension curve that is commonly used to introduce *cardiac* muscle dynamics in textbooks. Whether done for historical reasons (the skeletal L-T predates any cardiac one by 60 years) or for simplicity of explication, such use in instruction further encourages over-attributions of similarity between the two kinds of muscle. Furthermore, textbook demonstrations of a *cardiac* length-tension relationship of any kind are rare. Hence, students do not have an opportunity to examine the differences and to incorporate the implications of the differences into their thinking about heart function.

Implications. Two consequences result from these multiple influences. First, students mistakenly believe that cardiac muscle, like skeletal muscle, has a descending limb on its length-tension curve, and hence, that cardiac muscle can lose force by being stretched to these ranges. Second, the activational factors that are critical to the production of force in cardiac muscle (but less so in operational skeletal muscle) are further de-emphasized. However, everything that has been discussed so far has been with regard to individual muscle fibers. To affect students' thinking about heart failure, they must be extended by students to the intact heart.

Component 3: Whole-Heart (Collective) Muscle Function is the Same as Individual Fiber Function; The Frank-Starling Relationship is a Notational Variant of the Length-Tension Relationship (*Frank-Starling = Length-Tension*)

Background to component 3. If an intact ventricle of the heart is filled with progressively greater volumes of blood, it produces a progressively more forceful contraction. This relationship, given in Figure 12, is called the *Frank-Starling relationship*, after the two individuals who simultaneously established it. The Frank-Starling relationship (F-S) as presented in Figure 12 is, again, determined by laboratory procedures. The curve is sometimes presented in textbooks with a small descending limb at large volumes (e.g., Katz, 1977, p. 204), but with no explanation of why force declines at these laboratory-induced, forced volumes. (Force declines because the heart muscle gets destroyed.) A caveat may (but often may *not*) follow that such volumes do not occur in the *in vivo* heart (Katz, 1977, p. 205).

[Insert Figure 12 about here.]

The misconception in component 3. The *misconception* is that the Frank-Starling relationship for an intact ventricle is a direct reflection of the length-tension curve operating on the individual muscle fibers which make up the intact heart. By this account, the increasing force that occurs with increasing volume, on the ascending limb and "plateau" of the Frank-Starling relationship, occurs because component individual fibers are stretched to lengths on the ascending limb and plateau of the length-tension relationship. Force production declines at large volumes in the intact heart because individual fibers are stretched to the descending limb of their length-tension curves, as discussed in the protocol from a medical student given in Figure 13.

[Insert Figure 13 about here.[

What's wrong? The direct mapping assumed by students, between volumes on the Frank-Starling curve and lengths on the length-tension curve, is erroneous. Part of this mismatch is due to the geometric relation between length (or radius) and volume; large volume changes can occur with minimal changes in length. Hence, the dynamic range of lengths of fibers in an intact heart is greatly restricted, compared to the lengths occurring across the range of the laboratory-determined length-tension curve. In fact, most of the phenomenon of the Frank-Starling relationship occurs at fiber lengths corresponding to the plateau of the length-tension curve. Furthermore, the *length-tension relationship* that students (and others) assume in this misconception is the classical *skeletal* one (see Component 2) for, as we have seen, the corresponding cardiac length-tension curve has no descending limb. The mechanisms by which force is *actually* increased with increasing volumes in the intact heart (the Frank-Starling relationship) are, again, activational.

Why Do Students Believe Component 3?

Students. Of the many learner factors that might contribute to this particular belief (Coulson, Feltovich & Spiro, 1986), two seem particularly germane (see Figure 14). First, the students are assuming that the parts of a system in some sense "add-up" in combination to account for the function of the *intact* system (the heart): The whole is equal to the sum of the parts (*insulation from synergistic effects*). This is an example of what we earlier referred to as an analytic *prefigurative* reduction.

Emergent properties that exist because of the structure and collectivity of the system (e.g., restrictions in the dynamic ranges of length, but also other effects such as hypertrophic adaptation--to be discussed later) are lost in this kind of analytic decomposition and attempt at additive reassembly. Second, even though the Frank-Starling relationship is not a collective counterpart of the length-tension relationship, there are enough enticing apparent similarities to lead students to treat them as at least analogical. Volume is *like* length in some respects (i.e., they are dimensions of *size*). Force (of a ventricle) is like tension. The respective curves for length-tension and Frank-Starling, showing the main relationships, look somewhat alike (especially if someone considers the *cardiac* length-tension relationship). Hence, it is easy to think of the Frank-Starling phenomenon as analogous to the length-tension phenomenon. The danger is that the detailed differences can be lost in the superficial analogy (*analogy treated as isomorphism*), as appears to be happening with the students. This is reinforced to the extent that activational contributions to force-production (the main basis for the Frank-Starling relationship) are neglected in favor of mechanical accounts, which, as we have argued, is encouraged from many sources.

[Insert Figure 14 about here.]

Teaching. Textbooks typically present the Frank-Starling relationship as a two-dimensional curve, with the activational component implicitly held at one value (underdimensioning), rather than the three-dimensional surface that would be required if the activational dimension were to be represented as a variable (e.g., Katz, 1977, p. 204). This common simplification is, perhaps, a concession to the two-dimensional medium, but it serves to further de-emphasize the activational component. In addition, textbook accounts of the Frank-Starling relationship that do not make clear what is reflective of the laboratory and why (as noted earlier regarding the small down-turn in the curve), versus what is representative of the *in vivo* heart, may also add to the problem (a form of *sanitizing*).

Implications. The implication for student thinking is that the Frank-Starling relationship for the intact ventricle is taken to be anatomically and mechanically based, in the same way that the length-tension relationship for individual muscle is taken to be. It is also mistakenly presumed that individual fibers in the functional heart can operate over the full range of lengths involved in the length-tension relationship for the sarcomere, including those corresponding to the ascending limb, plateau and descending limb.

Component 4: Heart Failure Results from the Overstretching of Muscle Fiber Filaments (*Heart Failure Results from Stretch*)

The misconception in component 4. Heart failure is thought to result from the heart becoming enlarged, stretching individual muscle units to lengths corresponding to those on the descending limb of the length-tension curve, and thus reducing the contractile force the ventricle can generate, as illustrated in the protocol of a medical student, given in Figure 15.

What's wrong? Many of the problems with this explanation have already been discussed, including the lack of a descending limb when the appropriate *cardiac* length-tension relationship is considered. Two other pertinent points will be addressed here. First, there are structural properties (Spotnitz & Sonnenblick, 1976) of the *intact* heart, including collagenous matrices, and also functional properties (Huntsman, Rondinone & Martyn, 1983) which prevent individual fibers from being stretched to lengths that would correspond to the descending limb of a length-tension curve. Second, an adaptive process of the heart, hypertrophy, intervenes when fibers approach overly long lengths, adding sarcomeres in series so that individual sarcomeres are returned to more normal lengths (Spotnitz & Sonnenblick, 1976). Both of these factors--the structural properties of fibers *in complex* and the hypertrophic adaptation process--exist at the level of the heart as an intact system, a level that is missed due to the analytic reduction that assumes that the system can be additively assembled from its parts.

[Insert Figure 15 about here.]

Why Do students Believe Component 4?

Students. The heart in failure does enlarge, a classic sign of heart failure (see Figure 16). The question is whether it fails because it gets large, or, more appropriately, whether it gets large because it fails. This superficial "correlation" between heart size and failure, we presume, encourages the inappropriate causal attribution, when the actual underlying causal mechanisms are not considered in detail (a reductive use of evidence: directional causality attributed to correlation). Furthermore, "bigness" (in the heart) and "long-length" (in a muscle fiber) share a common semantic dimension that encourages and supports the attribution, perhaps through cognitive activational spread (Anderson, 1983; Collins & Loftus, 1975), whenever either the large heart of heart failure or the length-tension relationship of muscle is considered. Also, the properties of the intact heart that prevent overstretch (collective *structure* of the heart, and the collective *function* of hypertrophy) are not likely to be accounted for well in thinking about heart failure if students assume that whole heart function can be assembled from its constituent components.

[Insert Figure 16 about here.]

Teaching. The enlarged heart of heart failure is highly salient in clinical teaching about heart failure. It is one of the classic clinical signs monitored to determine the presence and severity of heart failure. The salience of this feature, interacting with other related features and interpretations we have mentioned, may serve to reinforce the idea of failure as resulting from "stretch." In addition, for convenience and clarity of exposition, the interrelated processes of hypertrophic adaptation and heart failure are often taught in different sections of texts (*compartmentalizing*).

Implications. The implication for students' thinking is that heart failure is believed to result from stretching individual myocardial fibers to lengths at which, for anatomical/mechanical reasons, they cannot generate adequate force. The heart is mistakenly believed to fail because it "falls over the edge" of the Frank-Starling curve, onto a "descending limb" that is a direct reflection of the descending limb of the skeletal length-tension curve. Again, Sliding Filament Theory dynamics that account for the descending limb of the skeletal length-tension curve are overextended, this time to the functional heart.

Activational contributions that are in fact responsible are not sufficiently considered. This is *not* to say that students do not know anything about activational components in muscle function. When students are directed to activational dynamics in our studies of muscle activation and contraction, they often discuss aspects of these dynamics appropriately. The point here is that in considering *heart failure*, for many students these activational dynamics are not integrated into the process as they should be, and the inappropriate anatomical/mechanical account lingers in their thinking. It is worth noting that the lack of integration of anatomical and activational factors that was found in students' thinking about the length-tension relationship is recapitulated in their thinking about heart failure.

More than a Simple Chain of Reasoning? A Network

It is easy, in a way, to understand the seductive plausibility of the explanation of heart failure as resulting from stretch. Why does heart failure happen? The heart is made up of individual muscle fibers; there is a sense in which individual muscle fibers fail because of stretch; so why not the heart which is composed of fibers? Is this misconception simply the result of a faulty chain of reasoning of the following sort?

LENGTH-TENSION IS ANATOMICAL (STRETCH) CARDIAC L-T =
SKELETAL L-T FRANK-STARLING =
LENGTH-TENSION HEART FAILURE RESULTS FROM STRETCH

The skeletal length-tension relationship is anatomically (stretch) based; the cardiac L-T is like the skeletal one; the Frank-Starling relationship for the whole cardiac muscle is like the individual L-T;

therefore, the whole heart fails because the individual fibers become stretched. A reasoning chain such as this probably accounts in part for the misconception. But, that is not all there is to it. As we have seen, there are holes in the chain (e.g., the existence of a descending limb for the cardiac length-tension curve) that seem to get filled in from elsewhere to be consistent with the overall chain. In addition, why is the *overall* belief so powerful that it overrides contrary explanations from the students' main curricular textbook?

Our hypothesis is that bits and pieces of knowledge, in themselves sometimes partly correct, sometimes partly wrong in aspects, or sometimes absent in critical places, interact with each other to create large-scale and robust misconceptions. Such a structure of knowledge could be represented as an interactive network, where fragments of knowledge connect in complicated ways to mutually strengthen or weaken each other and to produce aggregate knowledge that is functionally different (in this case stronger) than the pieces (McClelland, Rumelhart & the PDP Research Group, 1986; Rumelhart, McClelland & the PDP Research Group, 1986; Waltz, 1985;).

A depiction of this kind of knowledge structure, representing aspects of the heart failure misconception, is given in Figure 17. Interactive and synergistic effects occur in this network in several ways, as illustrated in the following examples. One form of interactive effect involves *extension of attributes* among entities, as has already been discussed in this chapter. If two entities have many attributes in common, then the likelihood of their similarity on others is enhanced. For example, if skeletal and cardiac muscle are similar in many respects, then it is easier to believe inappropriately that they both have similar activational properties, that they both have a descending limb on their length-tension curves, and so on. A second interactive effect involves *reciprocation*. Belief in A makes it easier to believe in B, and *vice versa*. Belief that cardiac muscle has a descending limb on its length-tension curve makes it easier to believe that an *in vivo* ventricle would lose force for mechanical/anatomical reasons at large blood volumes, and that this would be reflected in a downturn in the Frank-Starling relationship at large volumes. In turn, belief that the Frank-Starling relationship has a downturn in force at large blood volumes that are naturally possible (a belief abetted in part by ambiguous textbook treatments of this matter, as we have noted) bolsters belief in a (nonexistent) descending limb for the *cardiac* length-tension curve (especially if one knows about the *skeletal* L-T relationship).

[Insert Figure 17 about here.]

Another network-wide effect involves *pervasive colorations*. Colorative effects occur because of the presence in many places within the network of aspects of knowledge that bear a "family resemblance" to each other. An example from the heart failure misconception is the aspect of *size*. It is present in the length-tension relationship through *length*, in the Frank-Starling relationship through *volume*, and in the *bigness* of the failing heart. The presence of a common semantic dimension of size in all facets of muscle function reinforces both the importance of *size* generally and misconceptions associated with it within the facets themselves. As *size* emerges progressively (from many sources) as a recurrent theme during the course of the development of the misconception, it comes to take on the ability to bolster belief in the misconception independently of the 'reasoning chain' and to support all constituent misconceptions related to size.

Why is the reasoning chain portrayed earlier in this section so seductively plausible? The answer, we believe, raises yet another type of synergistic effect--that of *structural corroboration*. The argument is alluring because it is so efficiently compact and tidy. But, this "good form," is achieved only because the ideas contained within the reasoning chain have already been misconceived in ways that all align toward internal coherence across the network, enabling the "syllogistic" overlay. The pervasive colorative effect of size across the whole network inlay adds to this structural integrity. Structural corroboration refers to the contribution to belief that devolves from multifaceted well-formedness, accomplished by the widespread alignment of elements within the network.

Finally, yet another way that interactive effects are manifested is in the fact that reasoning within the network can proceed in *multiple directions*. We have seen how an initial focus on the length-tension relationship can lead to the inappropriate account of heart failure. But, what if a person starts with a focus on heart failure itself and tries to decipher a cause? It is easy, especially if the contribution of activation is not recognized appropriately, to step backward and to find a plausible account in the descending limb of the length-tension relationship.

The structure we propose for the inappropriate conception of heart failure (and, perhaps, for the nature of complex ideas in general) is a connected, interactive, reciprocating network. Fragments, pieces and partial dimensions of knowledge feedback on each other in nonlinear, reciprocal ways. In the case of the present misconception, everywhere the network is "jiggled" it broadcasts dysfunctional influences. The result is a whole that is stronger than the sum of its parts. Various *simplifications* of complex phenomena play a large part in the acquisition and maintenance of the (in *this* case) dysfunctional structure. These include simplifications on the part of the learner, perhaps to aid some form of coherent comprehension; on the part of the teaching process, perhaps to ease students into sophisticated understanding; and on the part of laboratory science, perhaps within the incremental quest for understanding of complex phenomena (but where consumers of research, e.g., students, see and are affected only by the partial products of the overall quest, without access to the "big picture").

HARNESSING THE POWER OF RECIPROCATING NETWORKS: SOME INSTRUCTIONAL DIRECTIONS

If conceptual understanding of complex phenomena is like this--interactive networks of partial knowledge from many sources in experience--is it possible to take *advantage* of the power of these networks: to turn the power of mutual, reciprocal knowledge bolstering around, so that everywhere such networks are activated they broadcast influences toward more correct views? Further investigations of the nature of these networks and of the possibility of utilizing their dynamics beneficially in the educational process constitute some of the current directions of our work.

One focus is on finding ways to convey complex biomedical material tractably, without the oversimplifications that appear to contribute to error. The medical curriculum is dense, the ideas are difficult, and the pace is fast. All this promotes attempts at simplification--on the part of both learners and teachers who must cope with the pace. In simplifying, students can gain some level of coherent and satisfying understanding, teachers can "get through" the material, easily scorable tests can be built and graded, and so forth. There is probably a faith that simpler initial understandings can be built on progressively through the curriculum.

In some instances such incremental approaches to instruction can be effective. However, under certain conditions, such approaches are susceptible to hazards of the kind we have shown in this chapter. First, oversimplified initial versions of a concept can produce a false sense of understanding and abort the pursuit of deeper understanding. When a concept is especially complex and multifaceted, this problem can become more acute, because partial misunderstandings can reinforce each other. Second, instructional efforts to challenge and change a student's oversimplifed conception (to raise it to a higher level of sophistication) may fail. The student will minimize discrepancies with the simpler, cognitively satisfying model so that this model can be retained. The student may not notice discrepant aspects of the concept or, if they are noticed, they may be filtered in interpretation toward the model that is already held. Local adjustments in this model will take precedence over fundamental reformulations. Third, educational strategies that attempt to teach complex concepts by focusing on their simpler components (with the hope of building toward fuller understanding) may encounter an additional kind of problem. Some concepts (and their components) are inherently inextricable from their organic functional context (as is the case in the phenomenon of heart failure). Concepts such as these involve, for example, synergistic properties or interactions among numerous variables. For concepts of this kind, any analytic decomposition misrepresents the concept fundamentally. One cannot make the components "add-up" to the whole, and there is no alternative in instruction but to

find ways to convey the irreducible complexity in a manner that is tractable. Fourth, as we have seen, incremental approaches that start with simplifications can engender associated habits of thought and learning that remain and interfere with advanced knowledge acquisition.

We are attempting to develop methods for making complex material tractable (Spiro et al., 1987; in press) in the instruction and learning of complex biomedical concepts, including the phenomenon of heart failure. One such approach involves the use of pedagogical elements that are themselves simple, but with an instructional guidance system that at all stages of learning forewarns about the elements' limitations and misleading aspects, and accentuates points of mutual embellishment among the elements. This approach will be discussed briefly in the context of the instructional use of analogies.

Instructional analogy can help to promote understanding but, as we have addressed elsewhere (Spiro et al., in press), when single analogies are applied to complex phenomena, they can also promote entrenched misunderstanding. Single analogies may miss aspects of a phenomenon and may mislead about others. We believe, for example, that part of the students' misunderstanding of heart failure results from an overreliance on the analogy of the "rowing crew" for the dynamics of subcellular force production (see Figure 18). This analogy promotes, for example, the idea that if the boat were somehow to shrink (with the same number of rowers), the oars could get tangled up, or be otherwise mechanically obstructed. This could, of course, contribute to the anatomical/mechanical explanation held by students (in opposition to the more appropriate activational account) for the ascending limb of the length-tension curve, and thus could ultimately contribute to the neglect of activational factors in the account of heart failure. The rowing crew analogy also implies that the cross-bridges of the sarcomere (the "oars") all act at the same time and in synchrony, further de-emphasizing elements of *activationally* based recruitment of cross-bridges (greater or fewer numbers act within any stroke, depending on the energetic activational level of the muscle).

We have designed an instructional method that utilizes multiple analogies for cardiac muscle function (Spiro et al., in press). Each analogy connects to others and reinforces their contributions on dimensions of muscle function that are mutually appropriate; or a new analogy fills in aspects missed by others; or it *punishes* aspects of others that are misleading. For example, an analogy of a "galley ship" (Figure 19) is used to counteract the effects of the rowing crew analogy with regard to synchrony and to convey aspects of activationally based variations in force production due to recruitment. These three kinds of connections (mutual embellishment, argumentation, punishment) among the members of the set of analogies can be used to form a network (cf. Hinton & McClelland, Rumelhart, 1986) of analogies that we believe will take advantage of the power of reciprocating networks to promote robust and functionally appropriate belief.

[Insert Figures 18 & 19 about here.]

In similar fashion, we are investigating other methods for harnessing the power of reciprocating cognitive networks to facilitate advanced knowledge acquisition for complex concepts. These efforts are guided by what we have learned about the potentially dysfunctional aspects of such networks.

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Authors Note

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Footnotes

[1]We thank Alan Lesgold for discussing this issue with us.

[2]Our method of studying students' understanding of a concept has two major components. One is a scheme for analyzing conceptual structure, which can be used to identify areas of potential cognitive difficulty. This scheme has both analytic and synthetic components. Because contributions to misunderstanding of a concept may occur because of errors in understanding of even its most basic elements, the analytic part involves breaking the concept into its primitive elements, typically concrete entities with physically realizable properties (e.g., definitions of "myosin" and "cross-bridges" with regard to cardiac muscle dynamics). The more synthetic aspect involves the combination of these elements according to higher-order relationships among them, and emergent, more abstract conceptual aspects not easily tied directly to the basic "building blocks."

For each concept studied, a probe set of discussion questions is created for use with students in the laboratory. These probe sets for a concept are closely tied to the conceptual analysis, and all probe sets have a similar general form. This form can be thought of as an hour glass, which starts general and open-ended, comes down to specific questions, definitional elements, etc., and then builds back up again to conceptual component combinations and clinical applications.

In particular, the first probe question is always the same and addresses analogies and other kinds of models a student may use in thinking about a concept. The second question is a full and open-ended discussion question that spans the entire concept of focus. This is included early in order to gain an appraisal of the student's understanding before any aid or prompting that may result from later items of the probe set. The discussion questions then come down to basic elements of a concept. The questions then expand into progressively higher aspects of the concept under focus. The final items of a probe set include carefully selected application questions, often including questions chosen to reveal classes of misconception that can be envisioned a *priori*.

These probe sets are used with medical students (and practitioners) in individual sessions in the laboratory. Students discuss each question, in order, for as long a time as they wish. This first pass through the set is observed by at least one project member, and there follows a period of directed questioning by the observer prompted from the student's discussions. At the conclusion of the session there follows a period of open discussion of the concept. A session for one concept (and student) usually lasts about 2 hours. The entire session is audio-taped for transcription.

Analyses are directed at various kinds of commonalties in responses, at patterns of interdependency among responses to probe set items that suggest coherent conceptual models, and at patterns of response across probe sets for a family of related concepts that serve to corroborate (or disconfirm) our ideas about a student's conceptual model or its aspects.

[3]We thank Dedre Gentner for suggesting this term for the phenomenon.

[4]This reduction will be recognized by the reader as resembling a cognitive operation that is commonly portrayed as useful, enabling prediction of attributes in the absence of specific evidence, or more generally contributing to cognitive efficiency ("default values" in "frames" or "schema" theory, e.g., Minsky, 1975; "default assignment" of missing values of an object based on its shared values with other objects in more recent PDP models, e.g., McClelland, Rumelhart & Hinton, 1986). The status of such operations as being common to people and as sometimes being serviceable *because* they enable a kind of efficiency is not at odds with our more negative treatment of them here. Our argument is that such generally efficient effects, while perhaps having a serviceable function in many simple and routine knowledge domains, may actually be a major hazard or *impediment* when it comes to learning difficult, complex, sometimes abstract, sometimes *counter-intuitive* ideas of the sort we are discussing. Such ideas are hard to understand partly *because* our mundane cognitive apparatus, honed in (one might

even say "designed for") the commonplace, is in many ways at odds with what is needed. One might argue that the more that ideas are "out of synch" with common cognitive mechanisms, the more difficult they will be.

[5]While such a treatment is not appropriate for this chapter, the reasons why this mechanical "overstretching" account of heart failure *cannot hold* are explicated in great detail in another paper (Coulson, Feltovich & Spiro, 1986).

[6]Students participated shortly after having had the most pertinent (cardiovascular) part of their curriculum in each year. These proportions are conservative since only clear statements of the misconception were counted.

Figure Captions

Figure 1. Schematic representation of the sarcomere.
Figure 2. Schematic representation of sarcomere during shortening.
Figure 3. Quote from a second-year medical student.
Figure 4. Quote from an established cardiovascular physician.
Figure 5. Quote from a reasonably current textbook of clinical cardiology (Goldberger, 1982).
Figure 6. Skeletal length-tension relationship.
Figure 7. Medical student discussing the length-tension relationship.
Figure 8. Why do students believe Component 1? Converging influences.
Figure 9. Medical student discussing the basis for the Frank-Starling, cardiac function relationship.
Figure 10. Cardiac length-tension relationship in comparison to skeletal length-tension relationship.
Figure 11. Why do students believe Component 2? Converging influences.
Figure 12. Frank-Starling relationship.
Figure 13. Medical student discussing the Frank-Starling relationship.
Figure 14. Why do students believe Component 3? Converging influences.
Figure 15. Medical student discussing heart failure.
Figure 16. Why do students believe Component 4? Converging influences.
Figure 17. The misconception; a network of reciprocating beliefs.
Figure 18. Effects of a single analogy, when it is used to represent or teach a complex phenomenon.
Figure 19. Recruitment of force producers: the galley ship analogy Multiple analogies can be used to augment single analogies, and to counteract their misleading aspects.

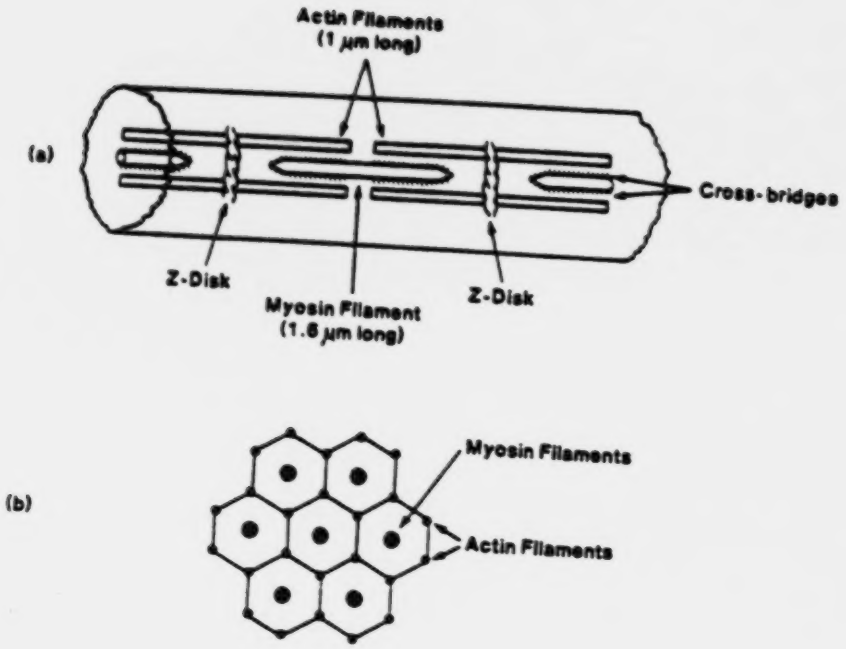

Actin Filaments
(1 μm long)
(a)
Cross-bridges
Z-Disk
Myosin Filament
(1.6 μm long)
Z-Disk
(b)
Myosin Filaments
Actin Filaments

Figure 1

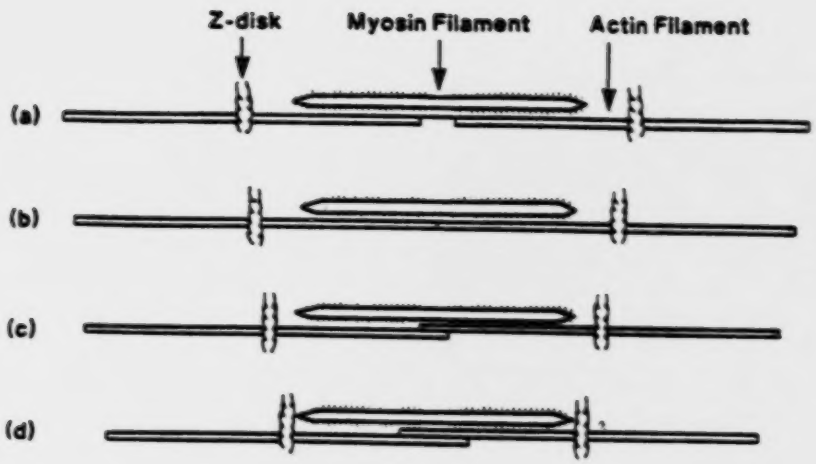


Figure 2

Okay, discuss factors which cause the muscle contraction to be inadequate (in heart failure). Okay, this takes me back to the Frank-Starling mechanism [Note: The Frank-Starling relationship describes how the heart produces greater force of contraction as it is filled with greater volumes of blood. This relationship and its role in the heart failure misconception are discussed in greater detail later.] where there's a volume overload on the heart and this, um, leads to, um, the muscle cell spindles to be spread apart so there's very little overlap between the actin and the myocin and ah, the way I conceptualize this is with the cross fibers as sort of like little guys rowing or else people pulling on a tug of rope, and ah, with less overlap they're able to ah, develop less tension and the entire muscle, muscle fiber itself, the heart is able to, is not able to do its job as effectively...I like to think of the Frank-Starling as falling over the edge of where, with, as the further the muscle cell gets stretched, the less it's able to do its job effectively...I use the sort of the, conceptualize the number of rowers or people pulling on a tug of rope to explain that with increasing stretching of each cell they are less able to pull and increasing stretching of each cell they are less able to pull and shorten. And also, in the Frank-Starling mechanism where it's gone over the edge of where more lengthening of the cell doesn't help with tension.

Figure 3

What the Frank-Starling relationship indicated was that ah, this ah, matter of ah, a muscle fiber being stretched, there comes a point at which ah, the stretching is no longer productive and, therefore, we tend to talk about the ah, healthy part of the curve, that's one phrase which is used commonly, which is the physiological ah, range that ah, as one stretches the muscle fiber, it will contract more forcefully. However, a point is reached ah, which is so-called plateau point where stretching the muscle fiber a little bit more is tolerated but does not result in any increased ah, contraction and force of contraction. And then if one stretches (the muscle fiber) a little bit further than that, we then go on to what is referred to as the down-slope of the curve where actually the ability to contract is decreased. The concept we have is that there is a point at which the left ventricle can be so dilated that it is no longer functioning.

Figure 4

Dilatation of the heart is also a factor that can decrease cardiac efficiency. The force of contraction of a muscle (including cardiac muscle) depends on the initial length of the muscle sarcomeres. When the sarcomere is initially stretched, this is associated with a more forceful contraction. The optimum sarcomere length is 2.2μm. At this length the overlapping between the actin and myosin filaments is ideally situated to allow the cross-bridges between them to pull the actin filaments inward during contraction. When the sarcomeres stretch beyond this point, the actin and myosin filaments do not overlap so much and the cross-bridges cannot pull the actin filaments inward adequately. As a result, the force of muscle contraction decreases. This is the structural basis for Starling's law of muscle contraction.

Figure 5

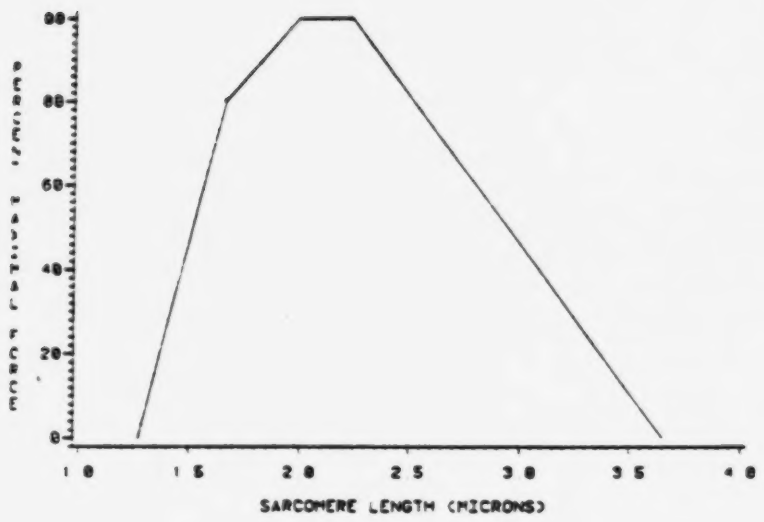
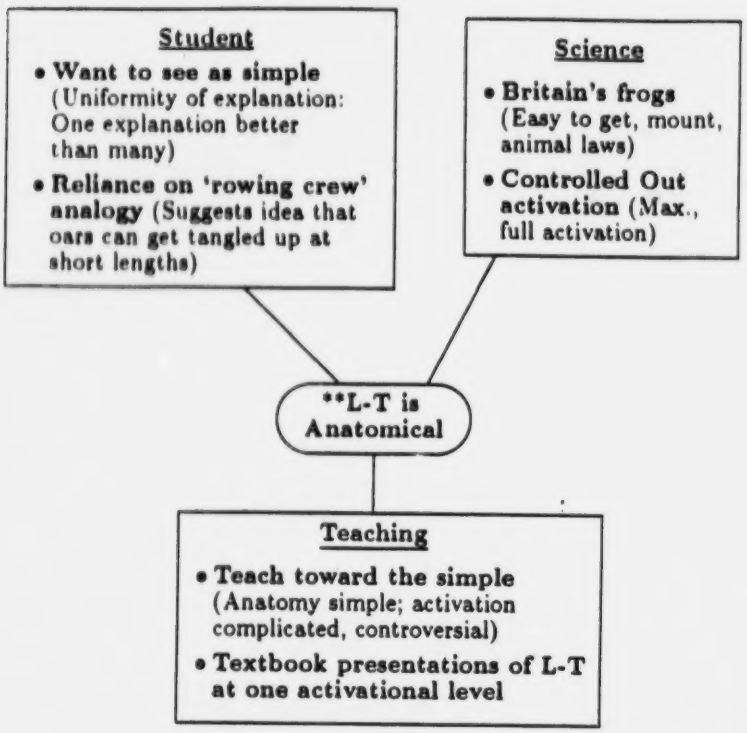


Figure 6

If you stretch any muscle excessively it's not going to be able to contract because you've pulled the actin and myosin completely apart, so that it can't form any cross-bridges and it can't contract, or, if you have a muscle that's too contracted to begin with, it can't contract anymore because the actin and myosin are pushed up against each other already and there's no room for them to slide any further.

Figure 7


Student
• Want to see as simple (Uniformity of explanation: One explanation better than many)
• Reliance on 'rowing crew' analogy (Suggests idea that oars can get tangled up at short lengths)
Science
• Britain's frogs (Easy to get, mount, animal laws)
• Controlled Out activation (Max., full activation)
**L-T is Anatomical
Teaching
• Teach toward the simple (Anatomy simple; activation complicated, controversial)
• Textbook presentations of L-T at one activational level

**Implication:

• Role of activation starts to get downplayed

Figure 8

Previously I was discussing the length-tension relationship and bringing into focus the idea that it is the length of contact between actin and myosin fibers (sic: actin and myosin compose filaments within fibers) which up to a certain point as the length increases will have a corresponding increase in tension upon contraction of that muscle fiber. However, after a certain point the length of actin-myosin contact decreases because the muscle fiber is stretched beyond a certain point. As a result, tension after that point decreases because the muscle fiber is stretched beyond a certain point. As a result, tension after that point decreases as the fiber gets stretched and pulled past a certain length...This is responsible for the fact that as end-diastolic volume is increased ah, and there is a corresponding increase in ah, contractile force and stroke volume at systole.

Figure 9

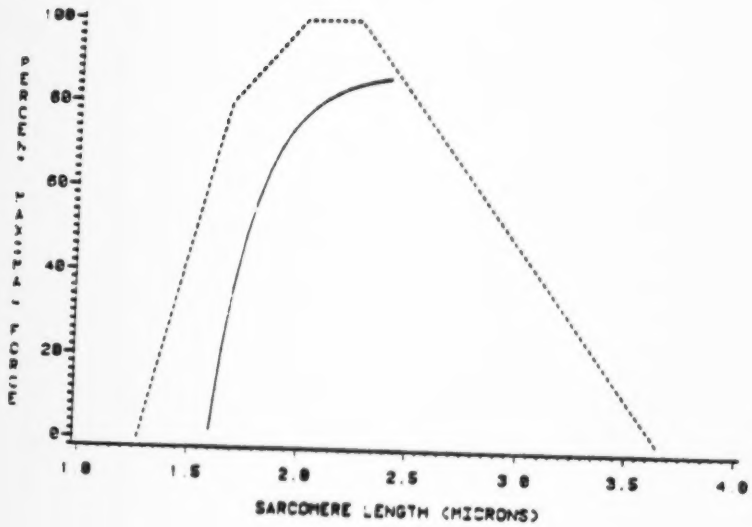
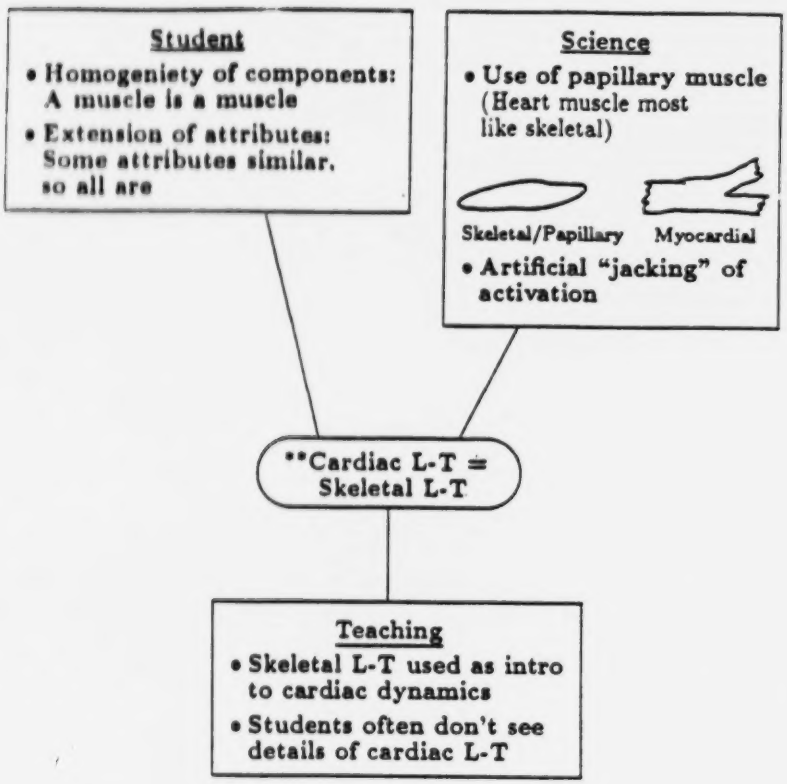

100
80
60
40
20
0
PERCENT MAXIMAL FORCE
1.0
1.5
2.0
2.5
3.0
3.5
4.0
SARCOMERE LENGTH (MICRONS)

Figure 10


Student
• Homogeniety of components: A muscle is a muscle
• Extension of attributes: Some attributes similar, so all are
Science
• Use of papillary muscle (Heart muscle most like skeletal)
Skeletal/Papillary
Myocardial
• Artificial "jacking" of activation
**Cardiac L-T = Skeletal L-T
Teaching
• Skeletal L-T used as intro to cardiac dynamics
• Students often don't see details of cardiac L-T

**Implications:

- Believe heart muscle has a descending limb
- Twitch activation not in picture

Figure 11

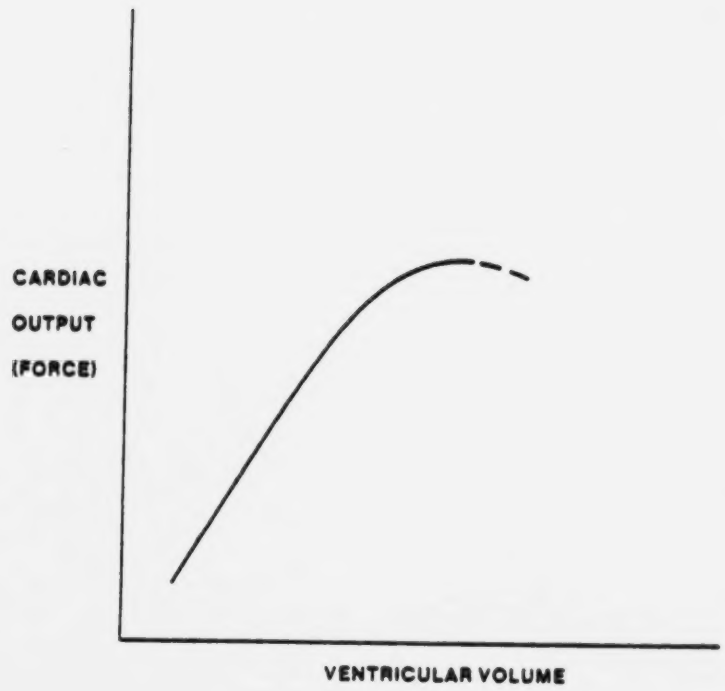
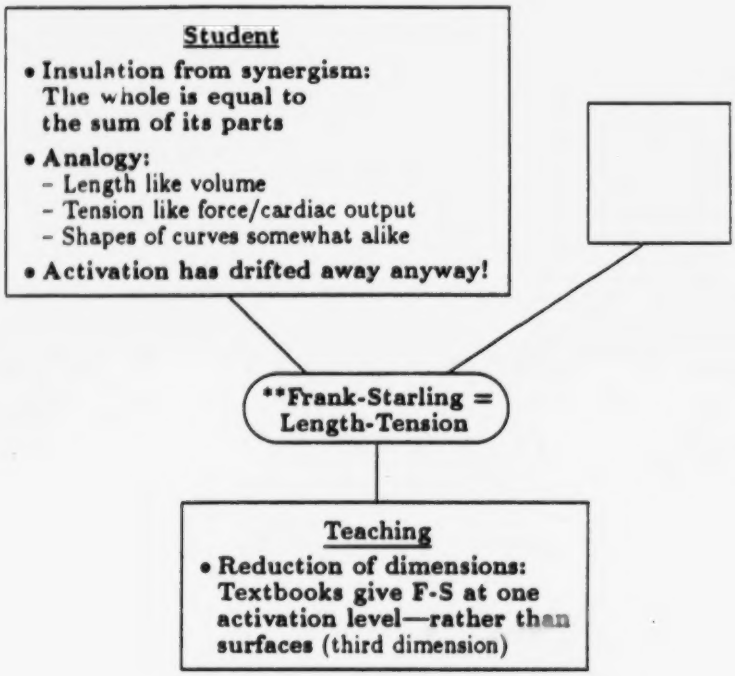


Figure 12

Starling's law states that as end-diastolic volume increases, the corresponding cardiac output at systole also increases. This occurs up to a point where the length of the fibers is too long and, therefore, tension at systole decreases causing a decrease in cardiac output. The decrease in contractile force corresponds with the length of myosin fibers being in contact with actin...as the length of contact between the fibers comes to a point where it starts to decrease because the cardiac muscle fiber is pulled to the length which is too long. That situation corresponds with the decrease in tension after a certain point is reached in the cardiac function curve.

Figure 13


Student
• Insulation from synergism: The whole is equal to the sum of its parts
• Analogy:
- Length like volume
- Tension like force/cardiac output
- Shapes of curves somewhat alike
• Activation has drifted away anyway!
**Frank-Starling = Length-Tension
Teaching
• Reduction of dimensions: Textbooks give F-S at one activation level—rather than surfaces (third dimension)

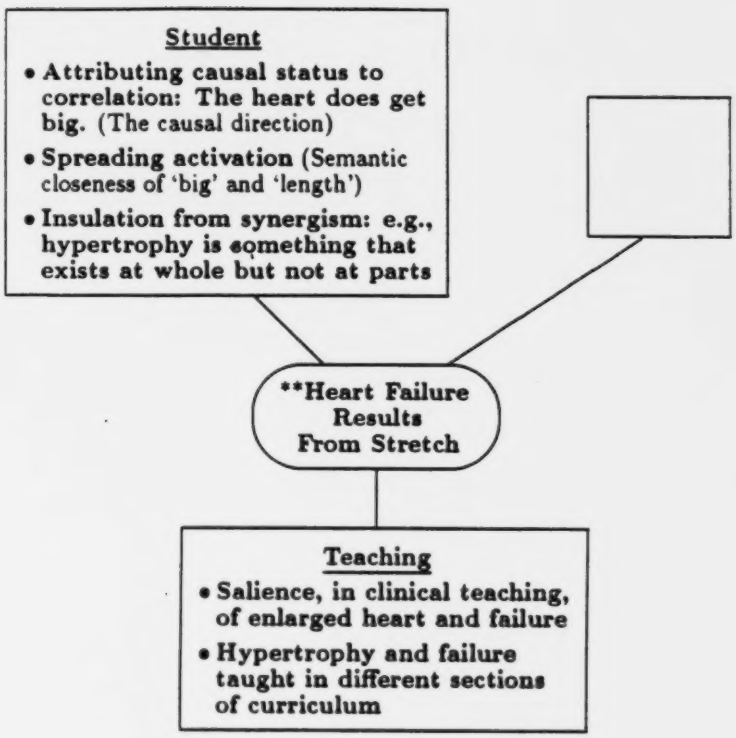
**Implication:

- The Frank-Starling relationship is believed to have an anatomical basis. In Frank-Starling, it is believed there are operative fiber lengths corresponding to the ascending, plateau, and descending limbs of the length-tension relationship.

Figure 14

Okay, the length-tension relationship of the muscle fiber...the length-tension curve. is a curve that relates to the sarcomere of the muscle fiber; as tension increases your length is going to increase (sic, backwards: for the active relationship being discussed, an increase in length results in an increase in tension). There's a plateau and then it drops towards the end...the cardiac function curve or the Frank-Starling relationship is what I was just talking about (above), the cardiac function curve goes up and then plateaus out...I'm just thinking of a curve in my mind where I see there's a certain level beyond which when it drops down it puts you into congestive heart failure.

Figure 15


Student
• Attributing causal status to correlation: The heart does get big. (The causal direction)
• Spreading activation (Semantic closeness of 'big' and 'length')
• Insulation from synergism: e.g., hypertrophy is something that exists at whole but not at parts
**Heart Failure Results From Stretch
Teaching
• Salience, in clinical teaching, of enlarged heart and failure
• Hypertrophy and failure taught in different sections of curriculum

**Implication:

- Heart failure results from stretch, has an anatomical basis

Figure 16

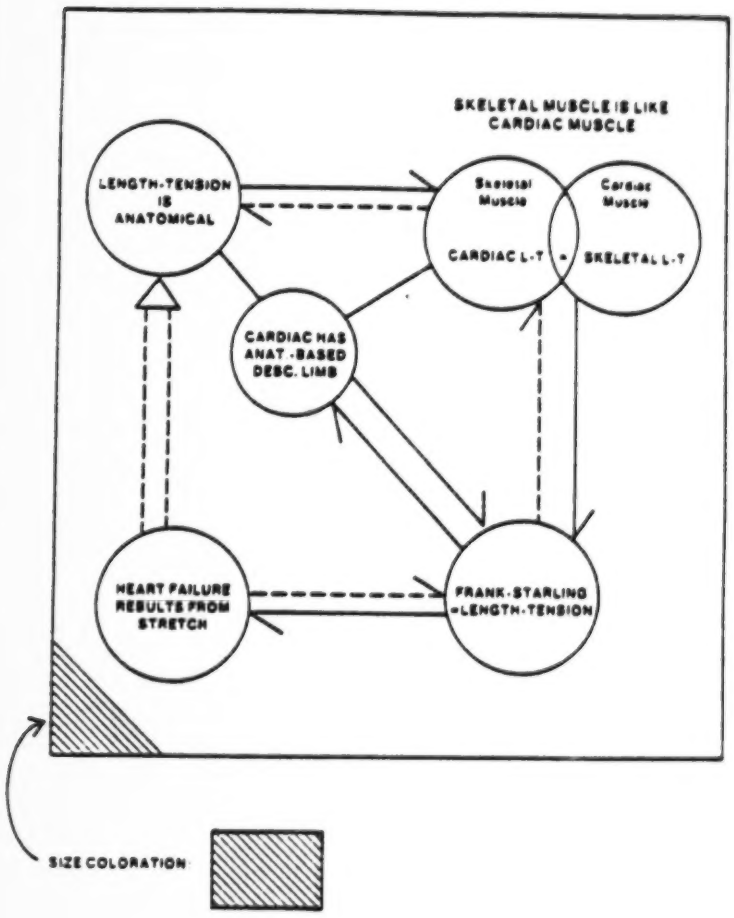
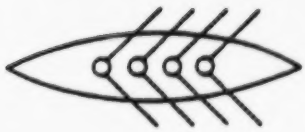

SKELETAL MUSCLE IS LIKE CARDIAC MUSCLE
LENGTH-TENSION IS ANATOMICAL
Skeletal Muscle
Cardiac Muscle
CARDIAC L-T
SKELETAL L-T
CARDIAC HAS ANAT.-BASED DESC. LIMB
HEART FAILURE RESULTS FROM STRETCH
FRANK-STARLING =LENGTH-TENSION
SIZE COLORATION

Figure 17



Rowing Crew

CAPTURES

(1) Anatomy of force producers: the "LITTLE ARMS"

(2) Nature of the movement of the force producers: Back and forth, hitting a resistance

(3) Lots of individual force producers

MISLEADS OR MISSES

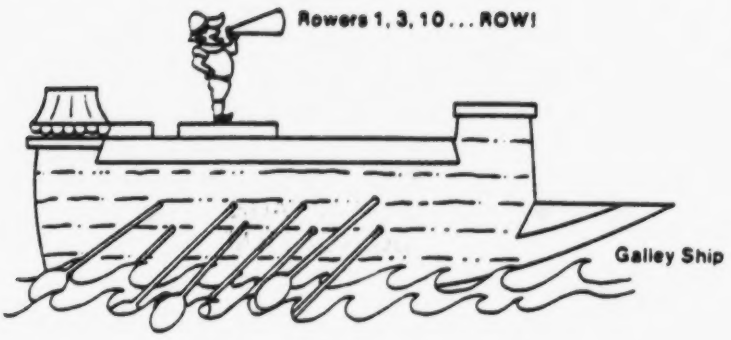
(1) Conveys synchronicity: idea that all producers act in unison

(2) Conveys notion that oars can get tangled (e.g., if boat too short)

(3) Misses actual nature of gross movement

(4) Misses things related to WIDTH

Figure 18



CAPTURES

(1) Control that selects and recruits which force producers are to work (on any stroke)

(2) Recruitment aspects of activation

MISSES

(1) Communication mechanisms

(2) Anything about internal metabolic, energetic, life processes of the force producers

Figure 19